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THE SURGICAL MANAGEMENT OF CHRONIC "SPONTANEOUS" PNEUMOTHORAX

REPORT ON ETIOLOGICAL FACTORS AND SURGICAL TREATMENT EMPLOYED IN FIFTEEN CASES

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INTRODUCTION

THE failure of the collapsed lung to re-expand after the occurrence of an attack of spontaneous pneumothorax results in chronic invalidism for the patient. In certain instances the persistence of the pneumothorax had been a major factor in contributing to the fatal outcome of these cases. Yet in spite of the potential seriousness of chronic or recurring pneumothorax, very little attention has been paid to this condition in the medical literature. There have not been many cases of chronic spontaneous pneumothorax reported in which active therapy has been administered. Most of these have been treated by chemical irritants. We have been able to find only 20 cases in the medical literature in which surgical measures have been employed,[1-12] 12 of which have been reported during the past year. Because the condition is primarily a mechanical one, we are convinced that in most instances surgical measures are indicated, if maximum pulmonary function is to be preserved. Certain general principles of surgical treatment have been presented in a previous communication by one of us.[13] This report is concerned with the discussion of the factors producing the spontaneous pneumothorax and the surgical therapy employed in a series of 15 patients treated during the past ten years.

TERMINOLOGY

The term, "spontaneous pneumothorax" is used to designate that type of pneumothorax coming on suddenly in seemingly healthy persons in whom there appears to be no obvious etiology for the pneumothorax. It occurs without external force and the disease processes responsible for this condition are generally

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asymptomatic at the time of the onset of the attack. Various other terms have been suggested, such as "idiopathic spontaneous pneumothorax," "pneumothorax simplex," "benign spontaneous pneumothorax," "spontaneous pneumothorax in the apparently healthy," etc. These terms are not in common usage. Since there is more than one condition causing the pneumothorax, it is difficult to coin a satisfactory descriptive word which would be all-inclusive. Although "spontaneous pneumothorax" does not imply the cause of the attack, it describes the pathologic state produced, and for want of a better term will probably be the one that is generally used.

On the other hand, "symptomatic" pneumothorax signifies that the pneumothorax occurs as a complication of an already recognized disease or known condition, such as pulmonary abscess, pulmonary tuberculosis, lung infarct, tumor, or trauma. At the onset of the attack the etiology of the pneumothorax is usually known. The management of this type of pneumothorax is directed mainly at the treatment of the particular underlying disease, and as such comprises a wider range of types of treatment which are not germane to the subject under consideration.

Our discussion is limited to the management of chronic and recurring pneumothorax occurring in apparently healthy persons. A spontaneous pneumothorax is generally considered chronic if the lung fails to expand in three months' time. In certain instances the time may be less than three months.

INCIDENCE

No reliable statistics are available on the true incidence of acute, chronic, or recurring spontaneous pneumothorax. This is due to the fact that the number of cases in the literature are few, and the fact that these patients are often diagnosed and treated as having a variety of other conditions. Since 1803 when Itard[14] first described this condition, only isolated cases have been presented for the most of this time. During the past two decades, however, a number of series from 50 to 100 cases have been reported from large clinics by Kjaergaard,[15] Perry,[16] Schneider and Reissman,[17] Leach,[18] and others. On studying the occurrence of spontaneous pneumothorax among college students, Blackford[19] found an approximate ratio of 1 case for every 1000 students at the University of Virginia. Thus, we find that cases of spontaneous pneumothorax are being diagnosed and reported with increasing frequency. In the great majority of patients with spontaneous pneumothorax the lung rapidly expands with rest and the adjustment of the pleural pressures. It is impossible to make an accurate estimate of the number of these cases in which the lung fails to respond. At the Los Angeles County Hospital, out of a series of 30 cases of spontaneous pneumothorax, only 1 became chronic, while 2 were recurrent. The other cases reported by us were private patients referred from various places, so they cannot be included in this figure. A chart (Table I) of some of the series in the literature shows the incidence of chronicity and recurrence of the pneumothorax to vary from 2 to 13 per cent.

TABLE I. INCIDENCE OF CHRONIC SPONTANEOUS PNEUMOTHORAX

SOURCE	CASES OF SPONTANEOUS PNEUMOTHORAX	CHRONIC	RECURRENT
Authors' series			
Los Angeles County Hospital	30	1 (31/3%)	2 (6%)
Kjaergaard[15]	51	7 (13%)	4 (7%)
Perry[16]	85	2 (2.3%)	4 (7%)
Total	166	10 (6%)	10 (6%)

The largest single series of cases in the literature has been reported by Brock.[12] Out of 71 cases, 46 were chronic while 25 were recurrent. In the authors' series all cases were chronic and 3 showed recurrent attacks. These figures do not represent an accurate picture of the incidence of this condition. Many of these cases are not accurately diagnosed, and are treated as pulmonary and pleural tuberculosis and other chronic diseases of the lungs. Also there is a tendency for physicians to believe that "the lung will expand in time" and no treatment is given. Such cases do not find their way into the literature.

ETIOLOGY OF SPONTANEOUS PNEUMOTHORAX

There has been considerable controversy in the literature over the cause of spontaneous pneumothorax, due to the fact that the roentgenogram usually fails to show the etiological lesion responsible for the collapse, and usually no further examination is made. In addition, only a limited number of patients have had careful studies, either at surgical operation or at autopsy. For this reason it has been frequently assumed that the normal lung and pleura could be torn by severe effort. The experimental observations of West[20] and Diez[21] have shown that it is impossible to develop sufficient intrapulmonary pressure to rupture the normal alveolus. If these observations are correct, we must assume the presence of some subpleural lesion or weakening of the alveolar wall, which will rupture with intrapleural pressures that can be developed by respiratory or muscular effort.

On direct observation of the pleural cavity in our series (13 open thoracotomy, 2 thoracoscopy) we were able to discover some sort of cyst, subpleural bleb, or bulla as the pathologic condition responsible for the spontaneous pneumothorax in 14 out of 15 cases. In the fifteenth case careful examination of the collapsed lung at open thoracotomy failed to show the cause of the pneumothorax (after adhesions binding it to the parietal pleura were freed). A ruptured cyst or bulla has been demonstrated in 87 out of 95 cases (combined medical literature and authors' series) in which direct observation of the lung has been made, either by thoracoscopy or open thoracotomy.

TABLE II. ETIOLOGY OF CHRONIC SPONTANEOUS PNEUMOTHORAX (AUTHORS' FIFTEEN CASES)

Ruptured congenital cyst	3	Ages 4 months, 6 and 17 years
Ruptured localized subpleural blebs or bullae	8	Ages 33, 30, 36, 31, 36, 36, 37, 50 years
Ruptured emphysematous bulla	3	Ages 38, 44, 45 years
No cause found	1	Age 32 years
Total	15	

Of course it is often impossible to find the tiny point of pulmonary rupture by thoracoscopy. We have found with direct observation of the lung at the time of the open thoracotomy it is most often necessary to submerge the lung under saline solution to observe the opening responsible for the pneumothorax. In 9 other cases in the literature of chronic spontaneous pneumothorax treated surgically where direct observation of the pleura was made, 8 showed some kind of bleb or cyst, while in 1 case no opening could be seen in the freed lung. Thus, the pathologic lesion actually observed as responsible for the production of the spontaneous pneumothorax in the vast majority of cases is a ruptured subpleural cyst, bleb, or bulla.

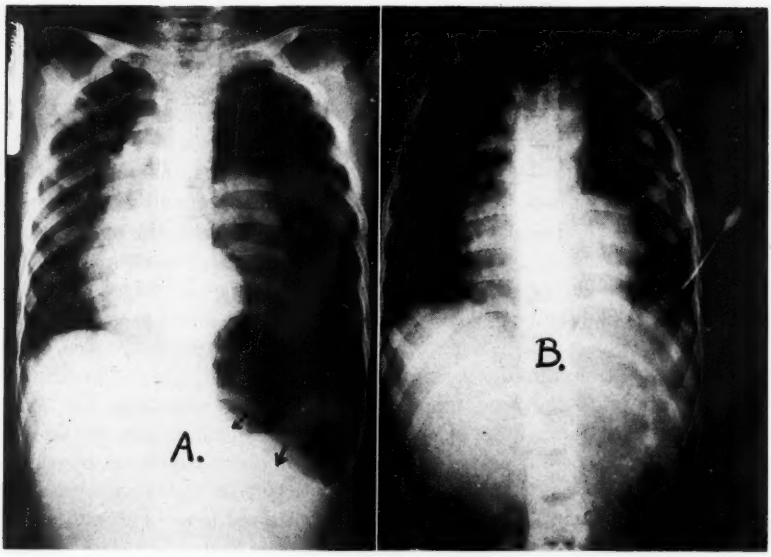
The medical literature is voluminous on the subject of the exact etiology of the formation of these thin-walled cysts, blebs, and bullae in the lung. For many years physicians have been fascinated by the discovery of these thin-walled pulmonary sacs, either by roentgen study or at post-mortem examination. It is not the purpose of this article to enter upon a detailed discussion of the theories of the development of these lesions. Certainly a few are congenital, but the majority are acquired. Rather, we arbitrarily classify them into three main groups in order to present a practical approach to the question. We realize that this classification may be open to criticism of being an oversimplification of the problem, yet we believe that it offers certain practical advantages in considering the management of these cases. The three main groups of thin-walled lesions responsible for spontaneous pneumothorax are: (1) congenital cysts, (2) localized acquired subpleural blebs or bullae, and (3) acquired blebs or bullae, part of a more generalized pulmonary emphysema.

Congenital cysts.—Three cases of congenital cysts were encountered in our series. Examples of this type of lesion are shown in Cases 1 and 2. Such lesions are seen commonly in the first two decades of life. They occurred in a 4-month-old infant and youths of 6 and 17 years in our series. Such congenital lesions represent a disturbance in development of the lung and have been frequently observed in newborn infants. The congenital cysts are single or multiple and are composed of a thin-walled sac lined by low cuboidal or pavement epithelium in which elements of the bronchial wall, such as smooth muscle, mucous glands, or even ciliated epithelium may be present. The opening with the bronchial tree is large, often being a branch bronchus. It is logical that the rupture of such a cyst would tend to result in a chronic pneumothorax because of the size of the bronchial opening and the fact that the cyst wall contains elements of the bronchial tissue. Clinically it may be difficult to differentiate between congenital and acquired cysts. Congenital cysts occur in younger persons and tend to be large. They more often contain fluid and are more apt to become secondarily infected. Congenital cysts are found in any portion of the lung. Localized acquired blebs or cysts are most commonly seen at the apex of the lung. The occurrence of the spontaneous pneumothorax in an infant or person in the first two decades of life should be considered as due to the rupture of a congenital cyst until proved otherwise. In most instances it is possible to distinguish between the congenital and acquired nature of the lesion only by

microscopic examination of the cyst wall. Because of the fact that the rupture of the congenital cyst does not tend to heal rapidly spontaneously, surgical extirpation of the cyst is indicated soon after the diagnosis is made. This is especially important in infants who tolerate a spontaneous pneumothorax poorly.

Localized Subpleural Blebs or Bullae.—Localized subpleural blebs or air vesicles have been reported as the etiological lesions found at post-mortem studies in persons in the second and third decades of life dying of spontaneous pneumothorax. They were observed in 8 of the cases in the authors' series as the lesion responsible for the pneumothorax. Examples of this type of lesion are seen in Cases 3, 4, and 5. We are indebted to the excellent pathologic studies of Hayashi,[22] Fischer,[23] and Kjaergaard[15] for a logical explanation of the development of these localized subpleural belbs or bullae. The subpleural blebs develop secondary to the production of a valvelike mechanism in the terminal bronchiole. Air may enter the alveolus freely on inspiration, but on expiration there is obstruction to the egress of air. The subpleural alveolus is gradually distended and with the stretching of the walls a bleb or vesicle is formed. The walls of the alveolus and visceral pleura are thinned out to one-half or one-eighth of the normal thickness.[24] The valvelike mechanism in the terminal bronchiole in most instances is the result of scarring of the lung and pleura, though in some cases no scar tissue has been observed. The simplest explanation of the formation of this scar tissue is that it develops from old healed subclinical pulmonary tuberculosis. The fibrosis, or a torsion on the terminal bronchiole, acts as a valvelike mechanism with trapping of the air and distention of the alveolus. It is significant that these localized subpleural vesicles are commonly seen at the apex, and are frequently associated with scarring of the pleura and intrapleural adhesions. Thus the blebs may develop as a result of mechanical changes produced by scar tissue of subclinical healed pulmonary tuberculosis. The appearance of these lesions in the second and third decades of life, when patients have usually been exposed to tuberculosis, is further evidence of the plausibility of this concept. The presence of scar tissue and trapping of air also explains why the aperture in the lung would tend to remain open.

Blebs or Bullae as Part of a More Generalized Emphysema.—Although pulmonary emphysema (overinflation and dilation of the pulmonary alveoli) has been extensively studied since the original description by Laennec[25] in 1819, there still exists considerable controversy as to the exact mechanism of development of this condition. On pathologic examination the alveoli are distended with a decrease or complete absence of elasticity. The walls between the alveoli are destroyed, so that several alveoli communicate to form one large air space. This condition is irreversible and the term chronic hypertrophic emphysema is applied. The alveoli may dilate on a functional or compensatory basis when there is contraction of a part of the lung by disease. These alveoli may return to normal, if the condition responsible for the contraction of pulmonary tissue is temporary. If it persists, then the compensatory emphysema may become



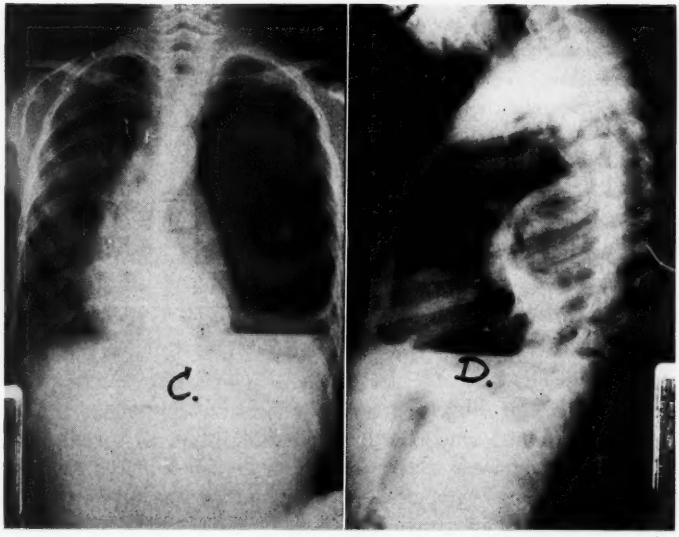
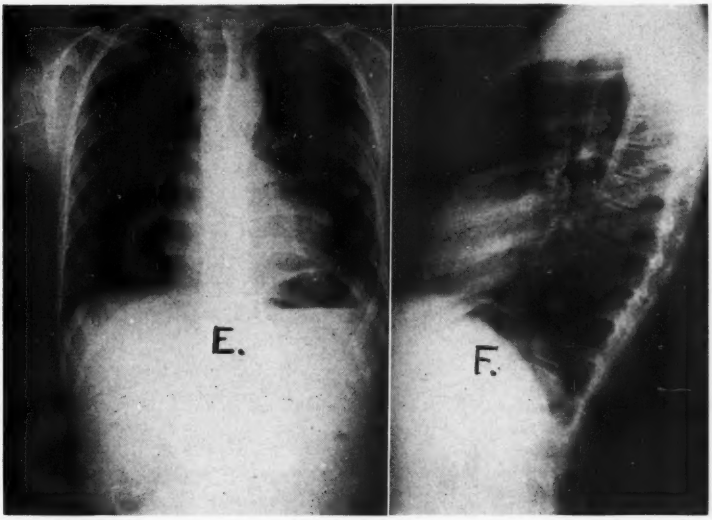
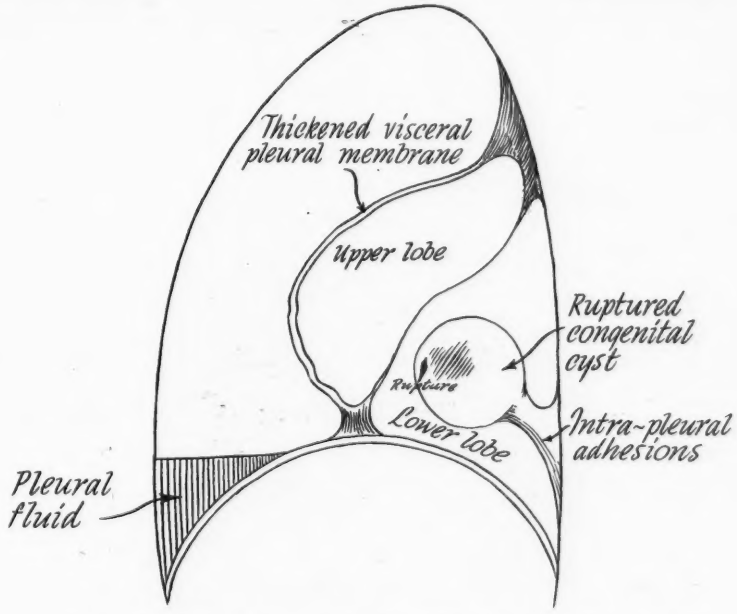


Fig. 1, *A* to *G* (Case 1).—A 6-year-old boy complained of dyspnea and pain in the left chest of six months' duration. In spite of repeated attempts to expand the lung, it remained chronically collapsed. Following open thoracotomy with resection of the ruptured congenital cyst, pneumonolysis of the lung, and pulmonary decortication, rapid expansion of the lung was obtained.

Fig. 1, *A* and *B*.—*A*, Roentgenogram taken immediately after onset of attack of spontaneous pneumothorax showing shift of mediastinum, depression of diaphragm, and dense adhesion between left lower lobe and chest wall. *B*, Temporary partial expansion of lung after insertion of needle attached to underwater drainage system.

Fig. 1, *C* and *D*.—Left lung 85 per cent collapsed with shift of mediastinum to the right. Compression changes right lung. Left lung compressed posteriorly on lateral film. Marked thickening of visceral pleural shadow. Fluid in left pleural cavity to the dome of the diaphragm.



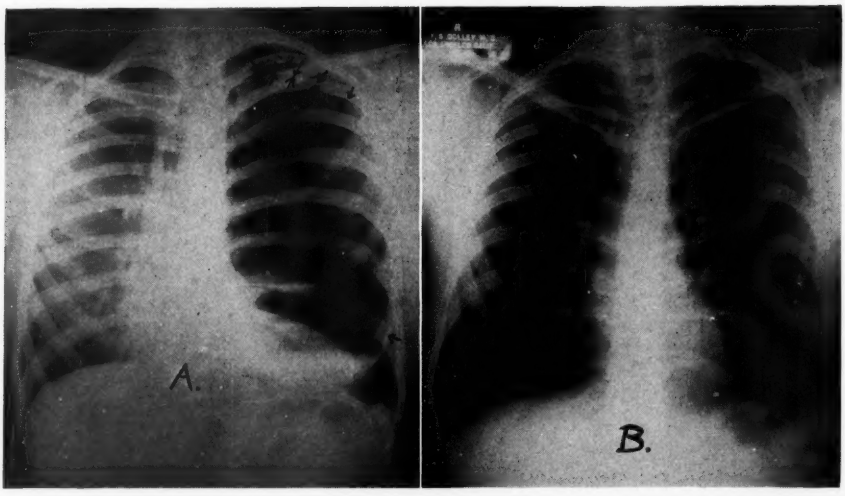


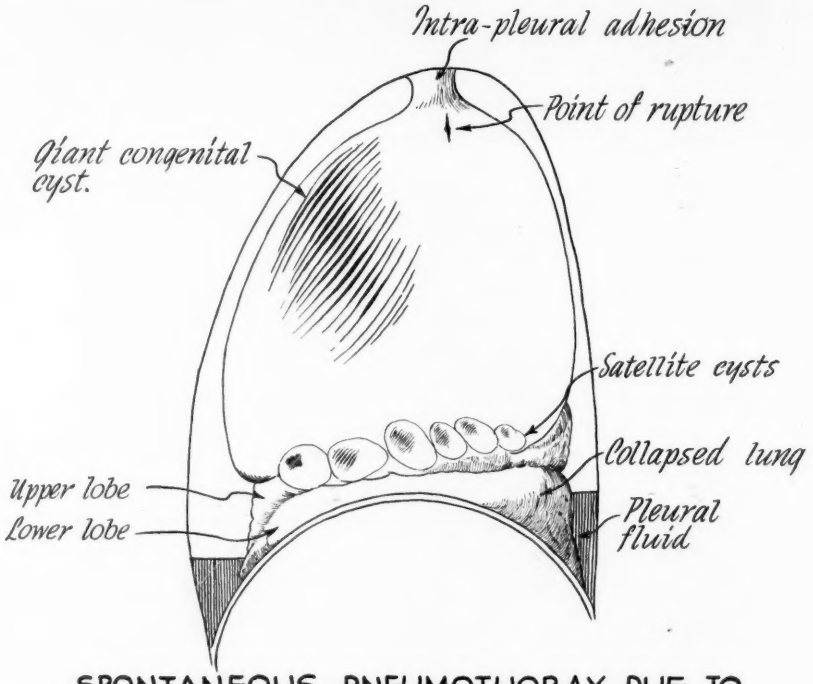
SPONTANEOUS PNEUMOTHORAX DUE TO RUPTURED CONGENITAL CYST.

G.

Fig. 1, *E* and *F*.—*E*, Posteroanterior view of chest two months following operation. The lung is completely expanded. *F*, Lateral view of chest. Note clearness of costophrenic angle. Fluoroscopy shows that the left diaphragm moves freely.

Fig. 1, *G*.—Drawing of the findings at operation. Lung greatly collapsed and held firmly by an inelastic visceral pleural membrane. Ruptured congenital cyst is held on tension by a dense intrapleural membrane. Pleural fluid is present. Rapid pulmonary expansion followed decortication of the lung, resection of the pulmonary cyst, and complete pneumonolysis.





SPONTANEOUS PNEUMOTHORAX DUE TO RUPTURED CONGENITAL CYST

C.

Fig. 2, *A* to *C* (Case 2).—A 17-year-old schoolboy had severe dyspnea for three months following an attack of left-sided pain. Attempts to expand the lung were unsuccessful. Weight loss of fifteen pounds.

Fig. 2, *A* and *B*.—*A*, Roentgenogram taken three months after onset of pneumothorax. Small pneumothorax left. Giant cyst of left upper lobe of lung held out by fine adhesions. Shift of the mediastinum to the right. Compression changes, right lung. Small amount of fluid left costophrenic angle. *B*, Film showing complete expansion of the lung. Patient well without recurrent attacks seven years after thoracotomy. He played tennis and has no physical impairment.

Fig. 2, *C*.—Small pneumothorax pocket present. A giant congenital cyst of left upper lobe is held up by dense adhesion at apex. Smaller satellite cysts are seen. Pleural fluid is present. Site of rupture of cyst demonstrated only on covering cyst with water. The lung expanded rapidly following the resection of the congenital cyst.

permanent. It is not our purpose to consider the many factors responsible for the production of emphysema. The most important are chronic or repeated pulmonary, bronchial, and bronchiolar infection, disturbance in the pulmonary circulation, and structural deformities of the chest wall and lung (Mayer and Rappaport[26]). With the destruction of pulmonary alveolar septa, and the thinning out of the alveolar walls, we have a "weakened point" in the lung. It would be expected that rupture of these bullae would be common. However,

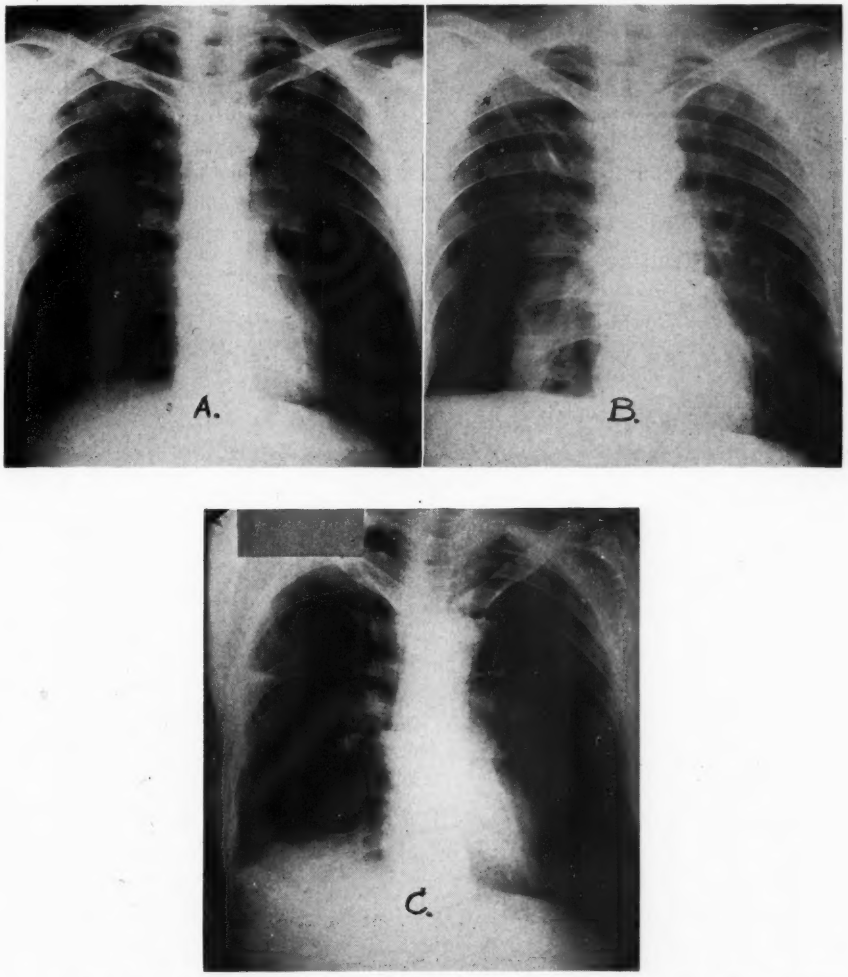


Fig. 3, *A* to *F* (Case 3).—A 31-year-old man had severe dyspnea and right chest pain for three months. The diagnosis of spontaneous pneumothorax was made and the right lung failed to expand on repeated aspirations. Patient was unable to work. Weight loss of fifteen pounds.

Fig. 3, *A* and *B*.—*A*, Routine roentgenogram taken six months before onset of spontaneous pneumothorax read as negative. *B*, Roentgenogram three months after occurrence of pneumothorax showing right lung 45 per cent collapsed. Fluid in left base. Extensive adhesions of right apex. Thickening of visceral pleural shadow.

Fig. 3, *C*.—Postoperative film showing expansion of right lung. Patient now well and working.

generally we do not have a valvelike mechanism, as there is usually a free communication with the bronchus. Thus, air is not trapped, and there is not as great tendency for these lesions to rupture as when a valvelike mechanism is present.

Four of the cases in our series showed evidence of pulmonary emphysema. Examples of this type of lesion are seen in Cases 6, 7, and 8. This finding was reported in 13 instances by Brock.[12] These cases are seen in an older age group. Our patients were 41, 44, 45, and 48 years of age. A history of previous repeated respiratory infection was given in all cases, which was not true of the second group. Some dyspnea on exertion had been present before the attack, but all of the patients were working and considered themselves only a little below par. In each of these cases large emphysematous bullae were seen, which were grossly similar to the more localized blebs. The intrapleural adhesions, seen in 3 of the cases near the bullae, may have exerted an abnormal tension on the bullae. Certainly, as will be pointed out later, they are an important factor in producing chronicity of the pneumothorax.

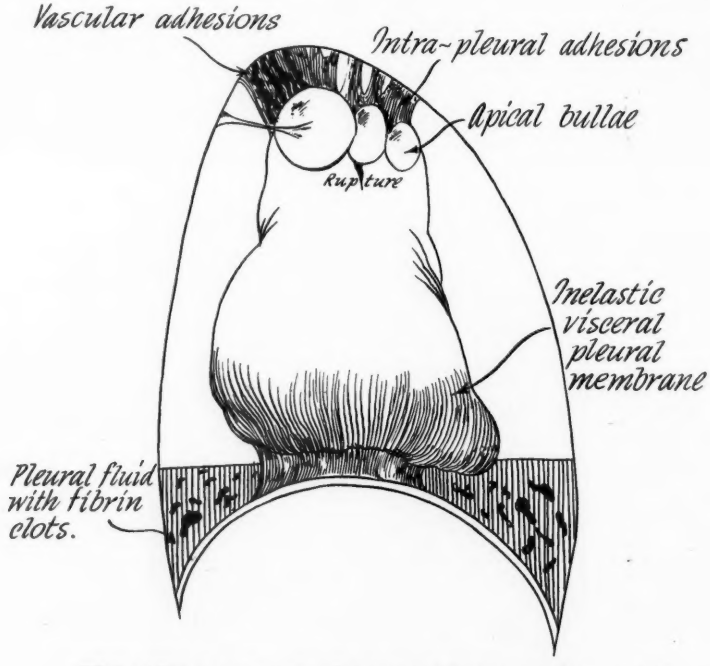
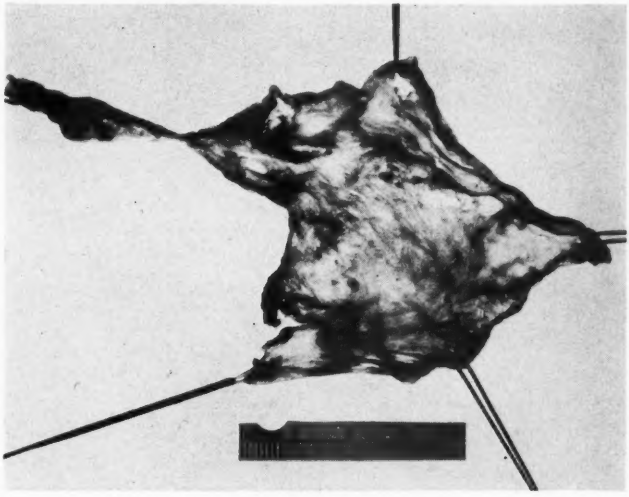


Fig. 3, *D*.—Drawing of right lung seen at operation. Right lung is 45 per cent collapsed and held captive by an inelastic visceral pleural membrane. Apical bullae are present with site of rupture at the base of one. Vascular adhesions hold lung at apex. Pleural fluid with fibrin clots is present. Resection of the bullae and decortication of the lung effected rapid pulmonary expansion.

CAUSES OF PERSISTENCE OF THE PNEUMOTHORAX

It is not always possible to tell exactly why the tiny rent in the wall of a bulla or cyst fails to heal, especially when there is complete collapse of the lung and mediastinal shift or herniation to the opposite side. Some of the openings are so small as to defy macroscopic identification without actually examining the

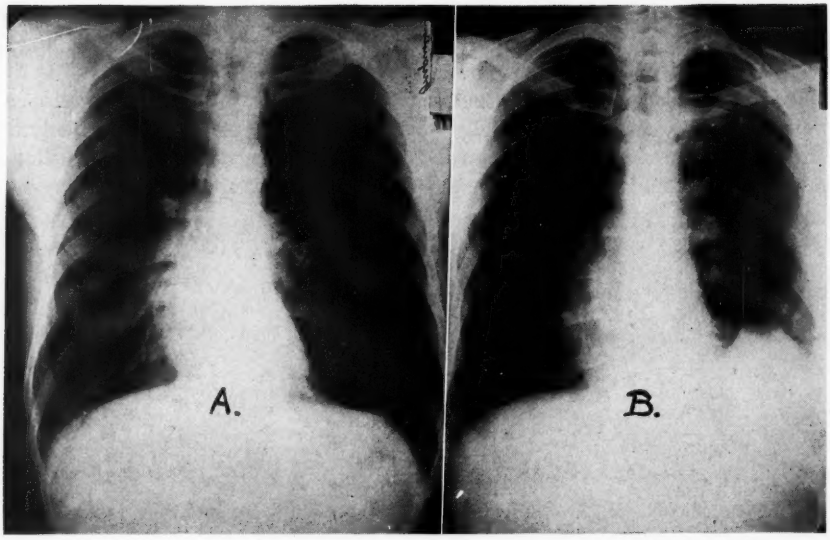


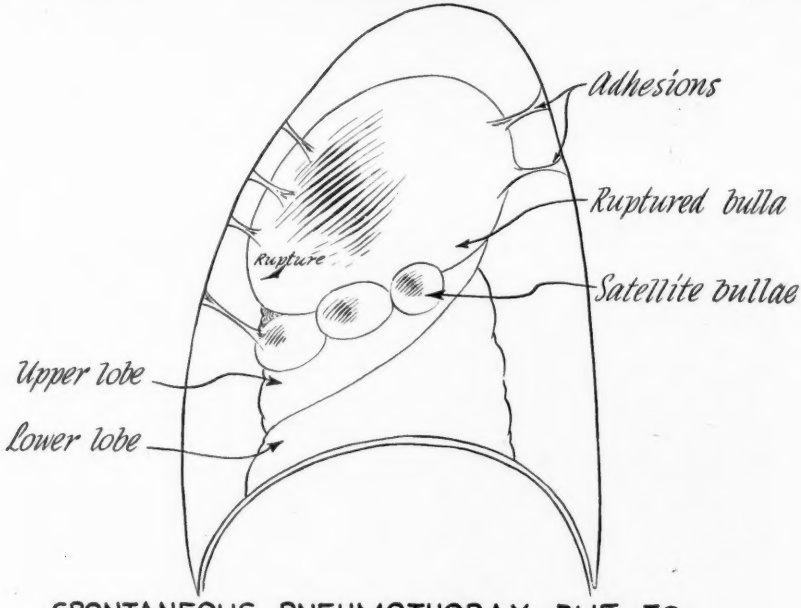
E.

F.

Fig. 3, *E.*—Gross specimen of part of fibroblastic membrane removed by decortication.

Fig. 3, *F.*—Photomicrograph of visceropleural membrane. The fibrous membrane is seen to be covered with mesothelium. A variety of inflammatory cells, foreign body giant cells, and capillaries is present.





SPONTANEOUS PNEUMOTHORAX DUE TO RUPTURED APICAL BULLA

C.

Fig. 4, *A* to *C* (Case 4).—A 36-year-old man was weak, tired, and short of breath following an attack of left chest pain three months previously. The attempts to expand the lung following the diagnosis of the pneumothorax had been unsuccessful. Patient had lost ten pounds, and had been confined to bed.

Fig. 4, *A* and *B*.—*A*, Roentgenogram three months after onset of pneumothorax. Left lung is 50 per cent collapsed. A suggestion of a large bulla is seen. *B*, Film six weeks after resection of the large bulla of left lung. The lung has completely expanded. Patient is now completely well.

Fig. 4, *C*.—Drawing of the findings at operation. Large apical ruptured bulla is held out by multiple adhesion. Several smaller bullae are present. Lung is 50 per cent collapsed. The lung expanded rapidly following pneumonolysis and resection of the bullae.

lung with the chest opened widely and testing for the aperture with saline solution and positive intrabronchial pressure. We recognize four factors as important in accounting for the failure of the lung to expand once an attack of spontaneous pneumothorax occurs: (1) intrapleural adhesions, (2) ruptured congenital cysts, (3) scar tissue in wall of bulla, bleb, or bronchiole, and (4) visceral pleural membrane.

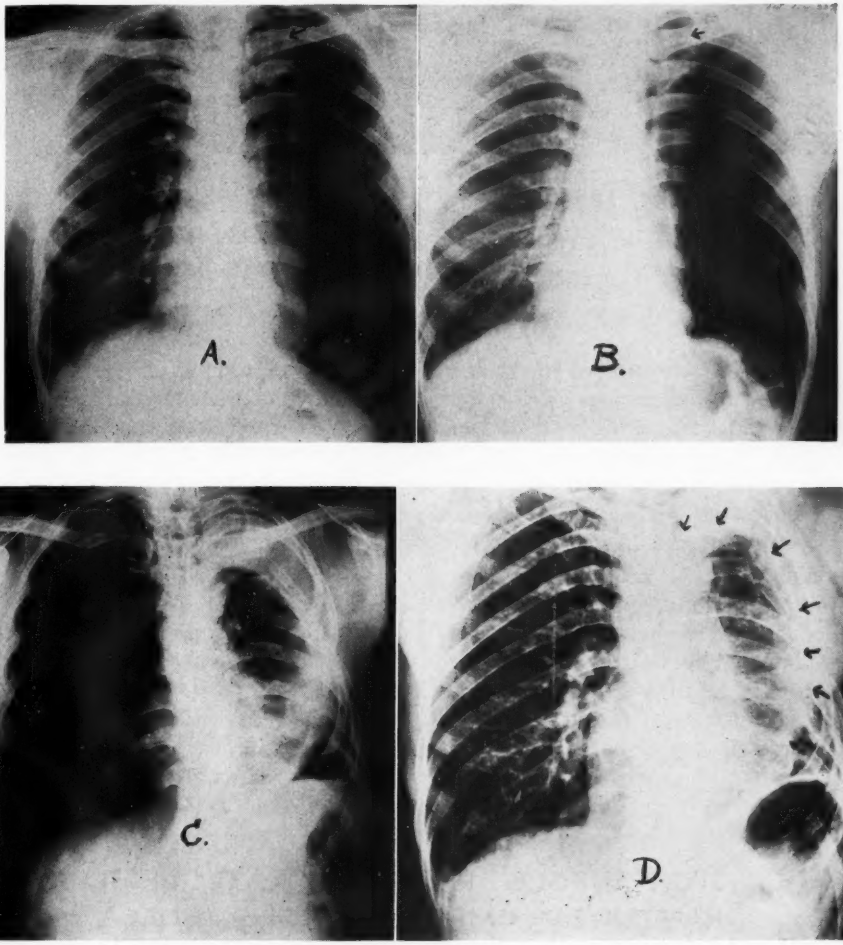
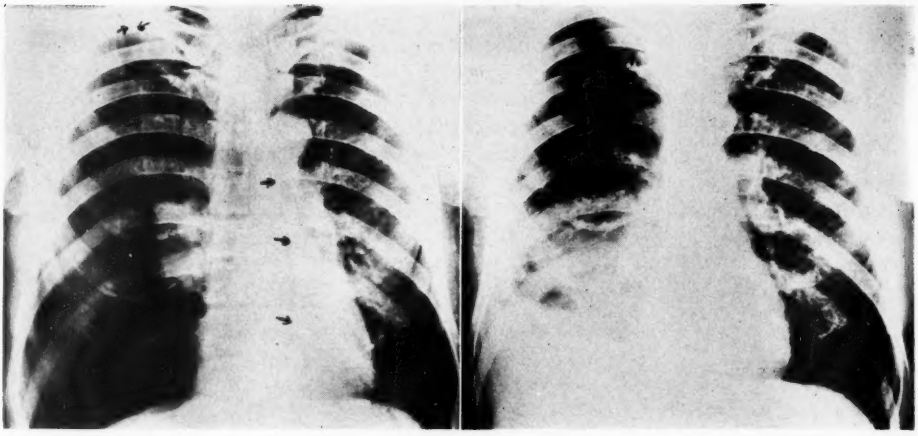


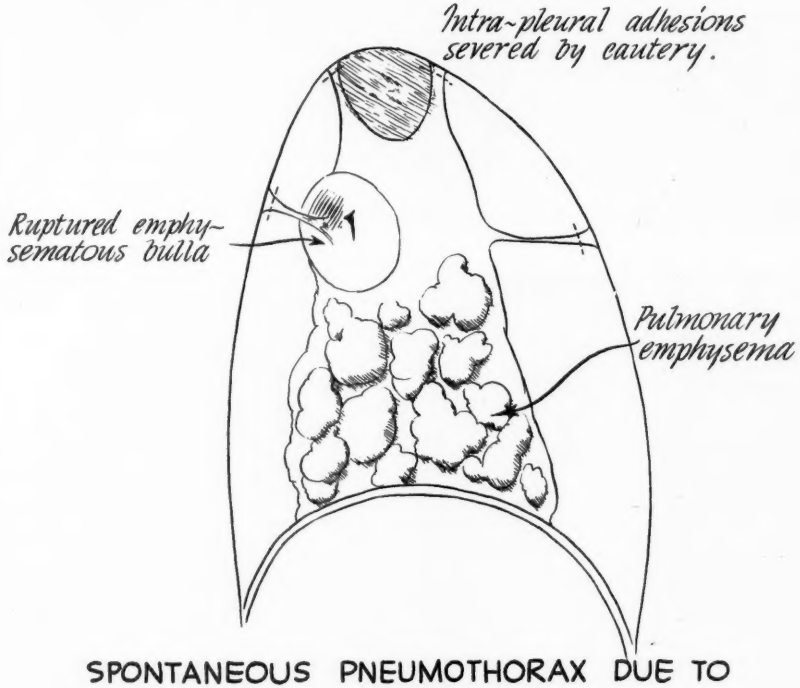
Fig. 5, *A* to *D* (Case 5).—A 37-year-old Japanese man had severe left chest pain and dyspnea for four months. Spontaneous pneumothorax was diagnosed and all measures to expand the lung had failed. Patient was a chronic invalid.

Fig. 5, *A* and *B*.—*A*, Pneumothorax left with 90 per cent collapse of lung, with intrapleural adhesions at apex of lung and diaphragm. Visceral pleura thickened. *B*, Following left phrenic paralysis no expansion of the lung. Thoracotomy then performed in 1941 with pneumonolysis and no decortication of lung, which we now believe was indicated in this case.

Fig. 5, *C* and *D*.—*C*, Two months after operation dense pleural shadow was present; no pneumothorax. Left diaphragm is high. *D*, Seven years later left pleurogenic fibrosis with shift of trachea and narrowing of intercostal spaces on the left. Left high frozen diaphragm. A decortication performed at time of pneumonolysis seven years previously would have prevented this.



A. *B.*



SPONTANEOUS PNEUMOTHORAX DUE TO RUPTURED EMPHYSEMATOUS BULLA

C.

Fig. 6, *A* to *C* (Case 6).—A 41-year-old man complained of slight cough and expectoration for years. Following an attack of right-sided chest pain, which had been proved to be due to a spontaneous pneumothorax on the right, he had been incapacitated. Attempts to re-expand the lung were unsuccessful. The lung expanded following division of adhesions by internal pneumonolysis.

Fig. 6, *A* and *B*.—*A*, Right lung 70 per cent collapsed with apex of lung held out by fine stringlike adhesions. A mediastinal hernia is present. The heart is shifted to the left. *B*, Roentgenogram taken two months after the pneumonolysis. Right lung has expanded. Bilateral emphysema is present. Patient is now well and working.

Fig. 6, *C*.—Drawing of right lung as seen at thoracoscopy. Ruptured emphysematous bulla is seen at right apex. Lung 70 per cent collapsed. Multiple adhesions holding apex on tension were severed with cautery. Emphysematous changes are seen in the remainder of the lung.

Intrapleural adhesions were present in 10 of our 15 cases. These adhesions prevent the lung from collapsing completely, so that there is tension on the lung, and the pulmonary rent is held open. In some instances these adhesions may exert a "guy wire" effect, resulting in a valvelike action at the site of rupture of the lung. Since the air is trapped in the pleural cavity, positive pressure may be built up to produce mediastinal shift or herniation. In addition to the high percentage of occurrence of these adhesions in our series, there have been enough cases of spontaneous pneumothorax reported in the literature showing intrapleural adhesions to emphasize the importance of this type of lesion in maintaining the pneumothorax. Severing of the adhesions in four instances in our cases allowed the lung to drop away from the chest wall and the mediastinum. The rent sealed over, and re-expansion of the lung was rapid.

TABLE III. CAUSES OF PERSISTENCE OF CHRONIC SPONTANEOUS PNEUMOTHORAX (AUTHORS' FIFTEEN CASES)

Pleural adhesions	10
Congenital cyst	3
Scar tissue in lung or bronchiole	5
Pleural membrane	7

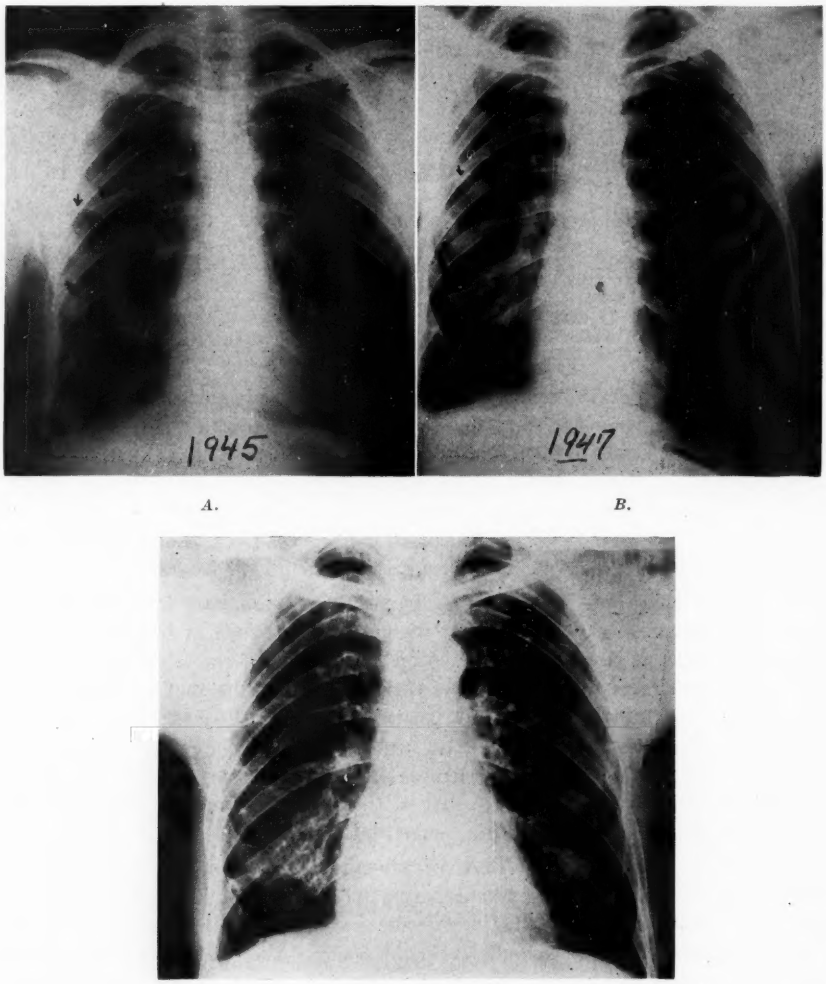
The structural characteristics of a congenital cyst are such that on rupture there is little tendency for spontaneous healing. The opening into the cyst is often a branch bronchus, lined by bronchial epithelium. The walls of the cyst are lined with cellular elements of the bronchial epithelium and bronchial walls. Since epithelial tissue does not seal over readily, the rapid healing of tears in the walls of a congenital cyst could not be expected. Thus, the very structure of congenital cysts tends to promote chronicity of the pneumothorax.

Localized blebs may be formed as the result of pulmonary and bronchiolar scars. Failure of these blebs to heal without the presence of intrapleural adhesions can perhaps be attributed to actual scar tissue in the lung or bronchiolar wall, which contracts to hold this aperture open. Yet the fact that the great majority of this type of bulla tend to heal spontaneously may mean that the valvelike mechanism and trapping of air does not persist after the lung is collapsed. When it does persist, a chronic pneumothorax may result. It is more difficult to understand why the rupture of these lesions results in chronic spontaneous pneumothorax than with the congenital type of cyst.

We have observed an additional phenomenon responsible for the persistence of the spontaneous pneumothorax, which has not been previously reported. A firm visceral pleural membrane was observed in 7 cases (6 of the 7 patients had surgery), to limit the expansion of the lung. This membrane may be formed on the visceral pleura as a result of the deposition of fibrin from the pleural effusion that occurs in some of these cases. When fibrin becomes organized, the lung is held captive in this tough envelope. In 1 case (Case 3) in which decortication was done, this membrane withstood a force of 1,500 Gm. without rupture. Fig. 3 *E* shows a photograph of a portion of the membrane which was decorticated from the collapsed lung in this case. Grossly it was

a tough, fibrous, gray-colored, opaque membrane, measuring from 1 to 3 mm. in thickness. Microscopic examination was reported* as follows:

Microscopic examination showed a membranous structure which consisted for the most part of fibrovascular tissue. The fibrous tissue varied from recently formed collagen to more compact scantily cellular collagen. The blood vessels



A. *B.*

C.

Fig. 7, *A* to *D* (Case 7).—A 38-year-old man had repeated attacks or left chest pain and dyspnea diagnosed as spontaneous pneumothorax for two years. For three months patient totally incapacitated because of persistent collapse of left lung. Weight loss of fifteen pounds.

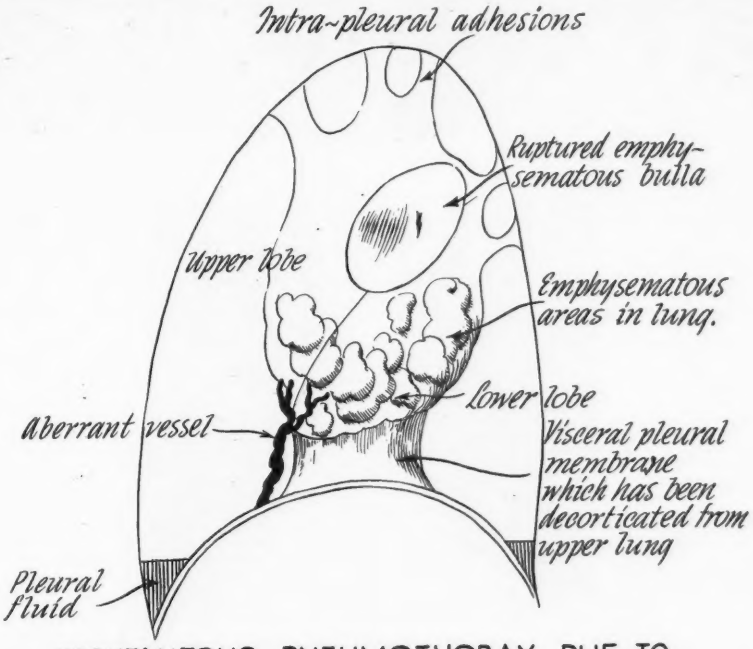
Fig. 7, *A* and *B*.—Chest film in 1945 shows small pneumonothorax left with large emphysematous bullae on right; 1947, increase in the pneumothorax on the left.

Fig. 7, *C*.—Postoperative film shows complete expansion of left lung. Bilateral emphysematous changes. Patient now well and working.

*By Dr. L. S. Tragerman, pathologist.

were mostly capillary channels. A few arterioles were seen. A scanty leucocytic infiltration was noted. This consisted mostly of lymphocytes and tended to occur about vascular channels. Some portions of the surface of the membrane were covered by fibrin, some of which appeared old and hyalinizing. Such surface areas frequently showed mesothelial lining cells, at times with swollen cytoplasm and prominent nuclei. Multinucleated giant cells of the general appearance of the foreign body type giant cell were also seen scattered near the surface of such fibrinous zones. No tubercles were seen. There was evidence of focal hemorrhage interpreted as part of the recent surgical procedure. Fig. 3 *F* is a microphotograph of a portion of this membrane. We have encountered no mention of this factor in the medical literature. Since almost one-half of our cases showed this condition, it must be a fairly common cause of chronicity of the spontaneous pneumothorax.

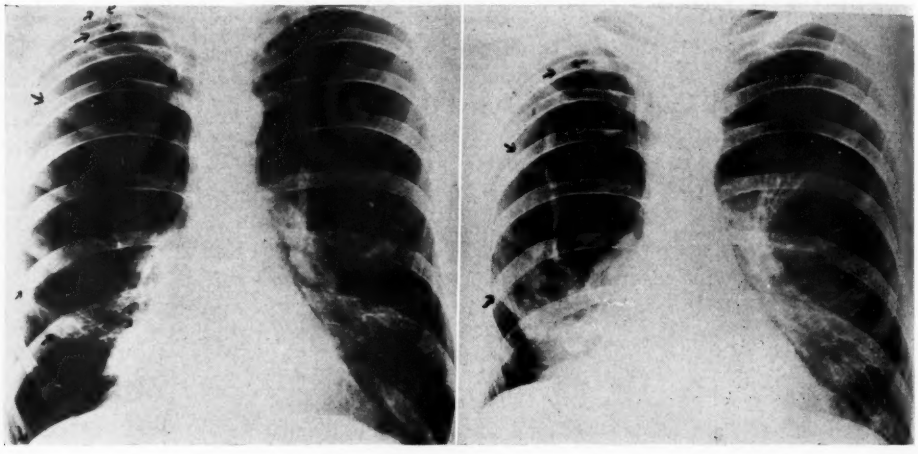
Before the war we were inclined to regard all deposits on the pleura as "thickened pleura," regardless of the etiology. As a result of the extensive wartime experience[27] with traumatic hemothorax, it has been learned that fibrin is deposited on the pleura in layers. After varying periods of time it becomes



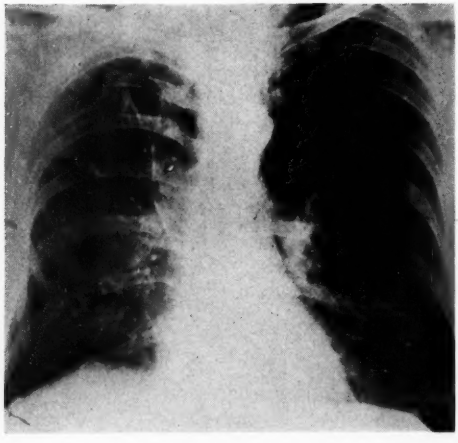
D.

Fig 7, *D.*—Drawing of findings at operation. Ruptured emphysematous bulla is present in subapical region. Emphysematous changes are seen in the rest of the lung. Lung is 50 per cent collapsed and held by inelastic visceral pleural membrane. Pleural fluid is present. Anomalous vessel extends from diaphragm to lower anterior lung. Pneumonolysis, pulmonary decortication, and resection of bulla effected. The lung expanded rapidly.

organized to form a true fibroblastic membrane. The length of time that this membrane remains as a separate layer, which may be peeled off the visceral pleura, depends upon the presence of infection in the pleural cavity and the subpleural lesion in the underlying lung. As has been pointed out, because the rupture of the lung is on a mechanical basis and not due to acute pulmonary infection beneath the pleura, infection in the pleural cavity has not been noted



A. *B.*



C.

Fig. 8, *A* to *D* (Case 8).—A 44-year-old man had chronic cough for years and mild dyspnea, but considered himself well. Three months before admission, following severe right-sided pain, patient had been diagnosed as having spontaneous pneumothorax. He was totally incapacitated and had lost twenty pounds.

Fig. 8, *A* and *B*.—*A*, Chest film showing pneumothorax right with complete collapse of right middle and lower lobes, and ill-defined huge cyst of upper lobe, held up by small adhesion. Generalized emphysema on left. *B*, Cyst of right upper lobe has collapsed more. Apical adhesion prominent. Thickening of visceral pleural shadow can be seen.

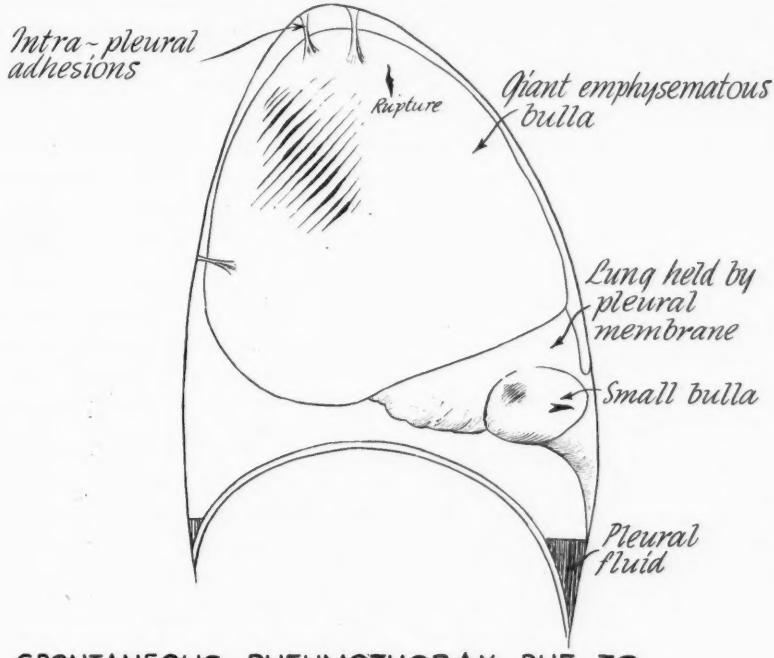
Fig. 8, *C*.—Postoperative film showing complete expansion of right lung. Bilateral emphysematous changes and fibrosis are present. Patient gained thirty pounds and is working steadily as an engineer.

in these cases. Thus the membrane does not become incorporated by fibroblastic proliferation as an integral part of the visceral pleura until several months have transpired, and it may be peeled off even after a period of five months.

DIAGNOSIS

The diagnosis of the acute attack of spontaneous pneumothorax is usually more difficult than that of the chronic state. The history of acute pain in chest or abdomen, dyspnea, prostration, and shock, coming on at rest or after moderate activity is well known. The clinical findings, leading to the performance of diagnostic roentgen examination, have been well emphasized in the literature[15-18] and need no repetition here.

In the chronic state, usually the diagnosis has been established before the patient sees the surgeon. The patient complains of dyspnea on slight exertion, weakness, and ease of fatigue. The appetite is poor and the patients are usually underweight. A history of some pain in the affected side of the chest usually



D.

Fig. 8, *D.*—Drawing of findings at operation. Small pneumothorax is present. A giant emphysematous bulla of the right upper lobe occupies most of the pleural cavity and is held out by intrapleural adhesions. Small point of rupture demonstrated near apex. Small satellite bulla is present on lower lobe. Pleural fluid present. Right lung markedly collapsed and held by inelastic pleural membrane. The two bullae were resected and the lung decorticated. Pulmonary expansion was rapid.

heralded the attack months before. On physical examination the patient may be either pale or of a dusky appearance, depending on the state of hypoxia that is present. Evidence of loss of weight is common, and breathlessness is seen with moderate exercise. The signs in the chest are those of fluid and air in the affected side and possible mediastinal shift. The affected side moves less and may actually be flattened. Râles indicating moisture in the bronchial tree or lungs are usually absent. The pulse is usually rapid after slight exercise, otherwise the cardiac findings are not noteworthy. We have not seen clubbing of the fingers in our cases.

On roentgen examination the pneumothorax is seen with varying amounts of fluid in the pleural cavity. In some instances the visceral pleural membrane shows up on the roentgenogram as a thickened pleural shadow. Usually it is impossible to detect the cause of the pneumothorax in the form of a bleb, bulla, or cyst of the collapsed lung by x-ray examination. The evidence of subpleural blebs or bullous emphysema on the affected or contralateral lung may be present. If a marked mediastinal shift is present, the contralateral lung may be compressed and show a mottled infiltration. In the differential diagnosis, it is important to rule out the presence of a so-called symptomatic pneumothorax. In most cases of the latter group the overt manifestations of the underlying disease are apparent. Careful history will usually elicit the symptoms of pulmonary suppuration or tumor, etc. Examination of the pleural fluid by smear and culture, and studies for tumor cells are indicated. Giant cysts of the lung may simulate a spontaneous pneumothorax. Evidence of trabeculae in the cysts or loculations of the cysts are important diagnostic aids. In some instances it is impossible to distinguish between a giant cyst and a spontaneous pneumothorax. We feel that in both instances surgical treatment is indicated. In one of our cases a giant congenital cyst held out by adhesions was present in addition to the pneumothorax (Case 2). In a second case a giant emphysematous bulla presented a similar finding (Case 8). Intrapleural pressure readings and the withdrawal of air in order to effect the expansion of the lung are indicated. However, one must proceed cautiously if a giant lung cyst is a possibility, particularly if the lesion is bilateral. The danger of puncturing a giant cyst wall and thus producing a pneumothorax is always present. We have done bronchoscopy in a few of these patients in which a bronchial block has been suspected or there was a productive cough. Bronchograms are indicated only when bronchiectasis is considered as being of clinical importance. All patients raising sputum should have a careful examination for tubercle bacilli by concentration and culture. In fact, the presence of cough and expectoration should warn the physician of a possible other diagnosis, or that he is dealing with a symptomatic pneumothorax. However, one of our patients with bullous emphysema did have an associated chronic bronchitis with cough and expectoration. With experience in treating these cases the diagnosis is usually obvious after a careful examination.

INDICATION FOR ACTIVE TREATMENT

We believe strongly that if the lung remains collapsed for a period of three months after an attack of spontaneous pneumothorax there is a definite indica-

tion for the institution of active therapy. The chances are exceedingly small that the lung will expand spontaneously after this period of time. In the past the inadequate treatment has been due partly to a lack of understanding of the factors responsible for the production and persistence of the pneumothorax. Failure to institute active treatment also stems from a belief that no active treatment is possible. Bed rest is important in the treatment of the acute spontaneous pneumothorax, but is entirely ineffectual in the chronic cases. The vogue for bed rest has been dictated by the idea that the lung will eventually expand and because the diagnosis of pulmonary or pleural tuberculosis has been made. As we have shown, both of these ideas are not well founded. However, the persistent pneumothorax may be just as disabling as pulmonary tuberculosis. Since the chest roentgenogram most often fails to demonstrate the presence of the etiological lesion in the expanded lung, we cannot be sure that a similar condition exists on the contralateral lung. Cases of bilateral spontaneous pneumothorax are being reported in the literature in increasing frequency. One of our patients, aged 23 years, developed a spontaneous pneumothorax of the left side following the successful resection of four pulmonary blebs for a spontaneous pneumothorax on the right. The fact that the right lung was successfully expanded following three years' pneumothorax, with the function of this lung preserved, certainly saved his life when the left lung suddenly collapsed. X-ray films failed to show any pathologic change in the left lung prior to the occurrence of the pneumothorax. The occurrence of a pneumonia in the uncollapsed lung may result in fatal outcome, as reported by Fisher.[28] We were called upon to see a patient in whom a chronic spontaneous pneumothorax had been present persistently for six years. Although a severe pleural reaction was present, the lung failed to expand. Because of dyspnea and chest pain, the patient was bedridden and had become a morphine addict. Attempts at expanding the lung were unsuccessful. He died of a coronary occlusion six years after the original attack of spontaneous pneumothorax. The impaired respiratory function and the hopeless outlook of the patient over a six-year period contributed to the coronary attack.

CHOICE OF TREATMENT

The two main forms of treatment have been employed to relieve chronic spontaneous pneumothorax: (1) artificial pleuritis by means of irritants introduced into the pleural cavity, and (2) surgical procedures.

Various types of irritants have been placed into the pleural cavity to set up a violent pleuritis, so that with the resultant pleural effusion and fibrin deposition the aperture in the visceral pleura and lung may be closed. A wide variety of pleural irritants have been used. These include Gomenol,[29] guaiacol,[30] iodoform,[30] iodized oil,[31] dextrose solutions,[32] silver nitrate,[12] iodized talc,[33] and others. The mode of employment and results have been well summarized by Brock,[12] and Hennell and Steinberg.[31] The number of patients treated by the production of a sterile pleuritis is now well over 100, while in only 31 (16 in the literature and 15 in our series) has surgical treatment been applied. The strongest argument for the use of pleural irritants may be found in the fact

that Brock,[12] who has reported the largest series in the literature, chose this means of treatment for all but 10 of his patients.

The popularity of the use of irritants to cause a violent pleuritis is open to considerable question. Having been through the early days of "poudrage," as a preliminary operation prior to lobectomy, we have had considerable experience with chemical pleuritis. Poudrage was the technique of Bethune, whereby iodized talc was insufflated into the pleural cavity to produce a sterile pleuritis. The adhesions thus formed over the upper lobe tended to prevent the collapse of this lobe, should a bronchial fistula develop in the lower lobe. The pleuritis so produced resulted in such a violent constitutional reaction that the patients were often sicker following this procedure than after the lobectomy. This procedure has been entirely abandoned as preliminary operation to lobectomy. The postoperative course of our patients following thoracotomy has been mild, and the average postoperative stay in the hospital has been one week. The postoperative morbidity compares very favorably with the chemical pleuritis where weeks and months may be required to expand the lung.

It has been repeatedly shown by bronchospirometry tests that pleural effusion resulting in chronic pleuritis with fibrosis markedly decreases pulmonary function. In these cases the main indication of treatment is to preserve the pulmonary function at the highest possible level because of the danger of bilateral disease. With surgical treatment, rapid expansion of the lung is accomplished with minimal pleuritis, because the mechanical defect has been corrected. Minimal pleuritis means maximum pulmonary function. Thus, surgery is preferred to preserve pulmonary function.

In 7 out of 16 cases (15 surgical, 1 nonsurgical) we have observed that a definite pleural membrane was present as the result of a pleuritis in which there was a persistence of the pneumothorax. In these cases the production of additional pleuritis and pleural thickening would seem to be useless. The lung has expanded promptly following operation, and no prolonged period of hospitalization is necessary. Since the lesion responsible for the pneumothorax presents a mechanical problem, correction of this mechanical defect is logically a matter of surgery. When dense adhesions hold the lung aperture open, the deposit of fibrin in the pleura might serve only to produce a limiting membrane preventing re-expansion. Examples of this result have been reported.[31] In spontaneous pneumothorax from congenital cysts or bullous emphysema, pleural irritants are not advisable. The subacute stage of a spontaneous pneumothorax produced by the rupture of small localized blebs would seem to be the most favorable indication for the use of these irritants. Then only those producing a mild pleural reaction should be used.

SURGICAL TREATMENT

The management of the acute form of spontaneous pneumothorax has been well covered in the medical literature[15, 16] and consists of rest and the adjustment of the pleural pressures to permit a gradual expansion of the lung. The surgical measures for the treatment of chronic spontaneous pneumothorax in-

clude: (1) closed catheter drainage, (2) phrenic paralysis, (3) thoracoscopy with internal pneumonolysis, and (4) thoracotomy with the employment of various surgical procedures.

Closed catheter drainage is a measure which is efficacious mainly for the acute and subacute forms of spontaneous pneumothorax. It is important that the catheter be introduced in the second intercostal space anteriorly so that fluid or the fibrin will not clog the tube. We include this method of treatment for the sake of completeness. It might be tried in certain poor-risk chronic cases in the absence of demonstrable adhesions, congenital cysts, pleural membrane, or where tension pneumothorax might develop.

Phrenic paralysis was first employed by Weiss[1] in 1930 to relax the diaphragm and decrease the tension on the lung in order to permit the pulmonary aperture to heal. In 2 cases in the literature this means was successful.[1, 2] We have attempted it in 1 case (Case 5, Fig. 5 *A* to *D*) with an unsuccessful result, because of the presence of mediastinal adhesions. We believe that phrenic paralysis is less desirable than more direct methods of treatment.

TABLE IV. TYPES OF SURGERY EMPLOYED
(AUTHORS' FIFTEEN CASES)

	CASES	SUCCESSFUL	FAILURE
Phrenic operation	1		1
Closed pneumonolysis	4	2	2
Thoracotomy:	13	13*	
With pneumonolysis	10		
With resection of cyst	10		
With decortication	5		

*One case lung expanded, but failure to do decortication resulted in marked pleurogenic fibrosis of lung.

Thoracoscopy is indicated as a diagnostic procedure and, when stringlike adhesions are present, as a therapeutic measure as well. Division of these adhesions with the cautery introduced through a thoracoscope was first successfully performed by Kipfer[3] in 1932, in order to relax the tension on the lung to permit rapid healing of the pulmonary rent. After 3 pneumonolyses, the lung expanded. We did thoracoscopy in 4 of our patients. In 2 instances we were able to divide cordlike adhesions with resultant successful expansion of the lung. In the other 2 cases the adhesions were too dense for division. Case 6 (Fig. 6 *A*, *B*, and *C*) shows the type of adhesions that may be divided by internal pneumonolysis. Brock thoracoscoped all of the patients in his large series, mainly for diagnostic reasons, but also as a means of producing artificial pleurodesis, by means of the introduction of silver nitrate. In 2 cases adhesions were successfully divided. Open thoracotomy offers a better visualization of the lesion, particularly when the exact location of the pulmonary aperture is not certain, unless the lung is covered with fluid and positive pressure administered. At one sitting the definitive surgical treatment can be employed with the greatest conservation of pulmonary function. For this reason we have preferred thoracotomy to thoracoscopy.

Formerly we reserved thoracotomy for those cases in which phrenic paralysis or closed pneumonolysis failed. Now we believe that it is indicated as a

primary procedure. It has been used successfully by us in 13 instances to effect a cure. The senior member of our group (F.S.D.) apparently was the first one to perform a thoracotomy to relieve a chronic pneumothorax in 1929. The pneumothorax occurred in a 3-month-old infant as the result of a lung perforation in a congenital cyst following the introduction of an aspiration needle. Complete collapse of the lung occurred and persisted for three months. At operation the tear in the lung was found and sutured. A tubular adhesion, still dangling from the chest wall, was excised. The baby made an uneventful recovery. From this early experience we have evolved the present-day techniques. The surgical procedures available with the chest open are: (1) a complete pneumonolysis, (2) surgical closure of the pulmonary opening, (3) resection of the pulmonary cyst or bulla, and (4) decortication of the lung.

A complete pneumonolysis may be safely performed through the open thorax without fear of hemorrhage. The lung is completely freed of tension and small lesions will close spontaneously. Pneumonolysis was performed in 10 cases, in 2 of which it was the sole intrathoracic surgical procedure necessary. In these 2 cases the aperture in the lung closed as soon as the tension was taken off the lung. In the other 8 cases, additional procedures were performed.

Surgical closure of the ruptured bleb or bullae by suturing has been performed in 2 instances.[6] We have not tried this as we believe resection of the affected area more effective and safe. In 1 of the cases in the literature[6] death occurred on the first postoperative day, either of mediastinal emphysema or a cerebral embolus.

Surgical resection of congenital cysts and large localized blebs and emphysematous bullae is the most direct approach to this problem. First reported by Sycamore in 1936,[5a] it has been performed 11 times in our series and 14 (5 local resections and 9 lobectomies) in the literature. Because we strongly believe that pulmonary tissue should be conserved, only the local or segmental resection of the pulmonary tissue should be employed. We have performed local resection in 10 cases, which included several extremely large cysts, and have not employed lobectomy in a single case. Lobectomy is to be avoided if possible, for the salvaging of even a few segments of a lobe may be important to the patient, if a contralateral pneumothorax occurs. Of the 9 cases of lobectomy reported in the literature (Brock, 3, and Hoyer and Clagett, 1), 1 patient succumbed to a contralateral spontaneous pneumothorax three months after operation. Examples of resection of the various types of cysts and bullae are reported in Cases 1, 2, 3, 4, 7, and 8.

As the result of extensive experience in World War II with the decortication of organized and infected hemothorax,[27] considerable speculation was voiced as to what the peace-time application of these lessons would be. In studying these cases of chronic spontaneous pneumothorax in light of our experience with battle casualties, we realized for the first time that the collapsed lung in these cases was held captive by a thickened fibroblastic pleural membrane in a manner similar to that observed in battle casualties with massive organized hemothorax. In fact, where such a pleural membrane exists, chemical pleurodesis and the surgical measures outlined previously will not be effective unless

this membrane can be removed. The techniques of decortication, standardized during World War II, are applicable here. We have found pleural membrane in 6 cases in this series.

We are surprised that this pathologic condition has not been described before, as there is no mention of this in the medical literature. In fact, an easily recognized pleural membrane (1 to 4 mm.), has been found in these cases. In one instance a tough, inelastic membrane withstood 1,500 Gm. of pressure without rupture (Case 3). The probable reason that this pleural membrane has not been described before is that only a few thoracotomies have been performed where the diagnosis could be accurately made. This condition may be only suspected by x-ray examination, and is easily overlooked on thoracoscopy. Actually we failed to appreciate the significance of such a membrane in the performance of an open thoracotomy for spontaneous pneumothorax in 1941, and failed to do decortication, which was definitely indicated (Case 5). Although the pleural opening sealed over, there developed a pleurogenic fibrosis with retraction of the chest wall, fixation of the diaphragm, and herniation of the upper mediastinum to the affected side. The effective pulmonary function of this lung must be very small indeed. Our experience with the rapid expansion of the lung following decortication in the 5 cases in which this procedure was done, and the minimal pleural thickening that follows this operation, leads us to believe that in Case 5 there would have been a great increase in the pulmonary function of the affected lung had we performed the decortication in 1941. Cases 1, 3, 7, and 8 present examples of combined decortication operation with resection of the cyst or bulla.

SURGICAL TECHNIQUES

Preoperative Preparation.—In preparing these patients for operation we have used the usual measures that are now commonly employed for pulmonary resection. In one instance where the pneumothorax was due to rupture of an emphysematous bulla (Case 8) the presence of a chronic productive cough necessitated several weeks' preparation with penicillin by intramuscular injection and aerosol route. Therapeutic bronchoscopy was helpful in improving the bronchial drainage (Case 8). In most instances the penicillin was administered only for several days before surgery and continued for two to three weeks after operation. Preoperative secondary anemia was amended by blood transfusion. Vitamin C and the B complex were administered in liberal quantities before and after operation.

Operative Technique.—The posterolateral approach was employed for the thoracotomy, using intratracheal anesthesia. In the younger individuals an intercostal incision was employed, while in older persons with a more rigid rib cage one rib was resected. After opening the pleural cavity, sterile saline solution was introduced into the pleural cavity to determine the source of the pulmonary rupture, much as one would examine a bicycle tire for a leak. With positive intrabronchial pressure administered by the anesthetist in all cases, except one in which we had freed the lung first, the site of the fistula was instantly obvious. Complete pneumonolysis was then done and the extent of the resection decided upon.

We have used simple resection of these peripheral lesions, clamping the lung and closing off the pulmonary tissue with mattress sutures of fine silk. In the case of large thin-walled cysts or bullae, it is well to open the cyst widely and proceed with the resection by keeping tension on the cyst wall. In this manner lung tissue may be conserved by the accurate identification of the attachment of the base of the cyst to the lung. It has not been necessary to do a segmental resection, though this procedure may be indicated, where the bleb lies close to the large pulmonary vessels. Actually we have dissected out the large pulmonary

vessels close to the bullae if we felt there was danger of catching the wall of these vessels with our suture. We have limited our resection to the lesions responsible for the pneumothorax and other peripheral large cysts or bullae which we felt might rupture at some future date. An attempt to remove all blebs or bullae is often technically impossible without pneumonectomy or lobectomy. We feel strongly that it is not only unnecessary, but is contraindicated if we are to conserve pulmonary tissue.

Decortication is performed if necessary, according to the standard techniques for clotted hemothorax.[27] We have found that the membrane peels off very easily. There has been very little oozing from the visceral pleura, which is shiny and glistening following the removal of the peel. The lung is carefully checked for bronchial fistula before closure. Tiny parenchymal bubbling, coming from the decorticated normal lung, is not serious and will be taken care of by the drainage tubes. Drainage tubes are inserted in the second or third intercostal spaces anteriorly and the sixth or seventh posteriorly, and are then connected immediately to a closed drainage system. The lung usually expands almost completely before the chest wall closure.

Postoperative Care.—The postoperative care is similar to that of a lobectomy, only the course is milder and the patients are not as sick. In most instances the patient feels the increase in breathing capacity as soon as he wakes up, and exclaims that he is using this lung for the first time in months. In only one instance was it necessary to aspirate tracheal secretions following operation. This was the case of a hypersensitive woman who refused to cough. Penicillin, in 100,000 units, is injected into the anterior drainage tube daily for two to three days, and the posterior tube is clamped for six hours after each injection. The drainage tubes are removed on the third or fourth days, as the lung has expanded and they cease to function.

COMPLICATIONS

There have been no complications in any of the adult patients incident to the surgical treatment employed. In a 3-week-old baby, the youngest patient in this series, a momentary cardiac standstill occurred during operation. This baby had spontaneous rupture of a congenital cyst soon after birth. In spite of closed catheter decompression of the pleural cavity, she remained in a precarious condition due to the large bronchial opening to the cyst. When we first saw her she was dehydrated, anemic, and in respiratory distress. After twenty-four hours' preparation we realized that the large fistula had to be closed if the baby was going to survive. The anesthesia was extremely difficult due to the baby's poor condition. A transitory cardiac arrest occurred, which responded to massage of the heart and the injection of 1/200 gr. of atropine sulfate. Following the operation she showed signs of cerebral malacia due to anoxia. These cleared in six months' time.

RESULTS

In all our series of patients there have been no recurrences and no deaths. All of the patients are leading fairly normal lives, as maximum pulmonary function has been preserved, with the exception of one (Case 5), where we failed to do a decortication. One patient, in whom a bulla was locally resected after collapse of this lung for almost three years, successfully withstood an attack of contralateral spontaneous pneumothorax, because most of the residual lung on the affected side had been preserved, and there was little pleuritis on the operated side. A number of the patients reported in our series have been operated upon during the past year, so we do not have long-time follow-up studies on these cases.

SUMMARY AND CONCLUSIONS

Chronic spontaneous pneumothorax is a potentially serious condition, as it leads to chronic invalidism which may be just as disabling as chronic pulmonary tuberculosis. Spontaneous pneumothorax is not "idiopathie" for, in the great majority of cases, the cause of the pneumothorax may be found in a ruptured subpleural cyst, bleb, or bulla. The etiology of these lesions is discussed. We recognize the important factors in the persistence of the pneumothorax as being the epithelial lining of congenital cysts, the presence of intrapleural adhesions, the scar tissue in the lung or bronchiole, and the formation of an inelastic visceropleural membrane. Since the pneumothorax persists because of mechanical conditions, surgery is indicated for their relief. The only other therapy has been the production of a pleurodesis by violent pleural irritant. We believe this method of therapy is definitely inferior to the surgical treatment presented, because of the limitation in pulmonary function so produced. Maximum pulmonary function should be preserved because of danger of a contralateral pneumothorax or other acute disease. The presence of the definite inelastic, fibroplastic, visceropleural membrane covering the lung is reported here for the first time in these cases, and the surgical importance of this lesion has been stressed. The technique of decortication of this membrane combined with pneumonolysis of the lung and resection of the ruptured pulmonary bulla in a series of 15 cases has been presented. The ideal procedure for the treatment of spontaneous pneumothorax is the one which will not only bring about obliteration of the pneumothorax pocket but also will return the best function of the lung. We believe that the surgical procedures outlined here will fulfill this ideal.

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DISCUSSION

DR. RICHARD H. MEADE, JR., Grand Rapids.—Ten patients with chronic spontaneous pneumothorax were treated during World War II at Kennedy General Hospital, and one in Chicago later. Our experience has run closely parallel to Dr. Brewer's. Of the 10 patients at Kennedy, 3 had bronchiogenic cysts, 1 of such extensive nature that a partial lobectomy had to be done; this man had some cysts in the lower lobe at the upper edge, evidence of cysts in the opposite lung, and some evidence of deformity of the renal pelvis. The two other cysts were easily removed without the necessity of doing a lobectomy. In 6 patients we could not find a definite etiological factor; all 6 had small openings in the lung which were sutured, but the chief procedure was decortication of the lung to allow re-expansion. I think this is the most important point, that the real cause of chronic pneumothorax is due to this enveloping membrane.

I should like to show a slide which illustrates very nicely the peel Dr. Brewer has just spoken of. In our experience it could not be removed from the lung as readily as in cases of chronic hemothorax or chronic empyema. The next slides illustrate the last case I had occasion to handle—a man who had extensive pneumothorax for several months. Before operating I decided to get pleural readings and to test the effect of continuous drainage; all I accomplished was the production of an extensive interstitial emphysema. He had such extensive pressure that with the needle in place, air escaped into the subcutaneous tissues. He was operated upon and it was found that he had multiple cysts, emphysematous cysts, involving the entire lung. We thought he also had emphysematous cysts in the opposite lung. It was obviously unwise to do a pneumonectomy and all we did was a decortication and enfolding of one of the cysts. Drainage was carried out and the tube connected with a water trap.

(Slide.) This was taken next day but you can see the extensive emphysema which was present. Only one tube was placed after the operation. That night it was necessary to introduce another tube in order to keep ahead of the air escaping from the pleural cavity. The next day he was "out of the woods" and the lung had begun to re-expand.

Five days later the lung had completely re-expanded and the tubes had been removed. (Slide.) This was taken a good many months later; he has continued to be well. I am sure he may have the same thing happen on the opposite side and he has been warned not to engage in too strenuous exercise.

This last case I think demonstrates the great value of closed drainage, whether using a water trap or active suction. I believe in most cases of chronic pneumothorax, even if it is impossible to demonstrate a definite lesion, if the lung is freed from its encapsulating membrane, and if closed drainage is applied, that will be all that is necessary.

DR. JULIAN MOORE, Asheville.—I would like to call attention to the danger of a hemorrhage in spontaneous pneumothorax. Ten years ago I saw one such hemorrhage which was fatal. The patient had a spontaneous pneumothorax; I saw him about twenty-four hours later and the hemorrhage continued in spite of repeated aspirations of blood and auto-transfusions. He was operated upon; one of the branches of the pulmonary artery in the upper lobe had been torn. The patient did not recover.

I have since seen 2 patients with hemorrhage associated with spontaneous pneumothorax. One of those patients recovered but was left with a fibrothorax because he was treated by late aspirations and was not submitted to surgery. Dr. Murphy and Dr. Elroy reported last year 1 patient who was seen three months after hemorrhage, and in whom decortication was successfully carried out and a good result obtained. About 50 cases have been reported in the literature in which spontaneous pneumothorax was associated with hemorrhage. If hemorrhage does occur, there is then the problem of controlling a serious hemorrhage as well as treating the pneumothorax.

DR. JOHN ALEXANDER, Ann Arbor.—I am discussing this paper to stress Dr. Brewer's warning against the indiscriminate injection into the pneumothorax space of glucose or blood or other irritating solution in an effort to seal the bronchopleural fistula. I realize that such solutions sometimes work well but, if they should fail to seal the fistula and if the lung should fail to expand, a thick membrane is likely to have been produced on both the visceral and parietal pleurae. This may permanently prevent the expansion of the lung and might prevent a subsequent successful decortication operation.

In our experience we have only infrequently found a pleural scar in patients having uncomplicated chronic spontaneous pneumothorax. I recall several patients whose chests have been incised, and who had had no evidence of a pleural scar or membrane; during the months before operation was finally accepted as necessary, there had been no evidence of pleural fluid. At the time of operation one or several ruptured blebs were found; the only steps needed at operation were (1) local excision of that part of the cortex of the lung that contained the blebs and (2) a gentle stroking of the entire visceral and pareital pleurae with dry gauze to promote complete obliteration of the pleural cavity after the lung had expanded.

After a wedge-shaped resection of a portion of the cortex, the sutures used to close the gap in the lung are under slight tension and might tear the lung if the pneumothorax air were rapidly evacuated by suction. I prefer to leave the lung partially collapsed while firm healing of the sutured area is taking place; absorption or intermittent aspiration of the pneumothorax air gently and gradually expands the lung. I am not sure, however, that this method is preferable to immediate expansion by suction drainage.

Dr. Blades has had great success from the local application of talc powder to areas in which bronchopleural fistulas are seen at the time of a thoracotomy.

DR. ARTHUR S. W. TOUROFF, New York.—I think that all of us are agreed that if the lung is collapsed and bound down by a fibrotic membrane, surgical treatment, namely, decortication, is indicated. However, I would like to say a word in defense of conservative

treatment of chronic pneumothorax of the usual variety. In that connection, I refer you to two articles by Dr. Herman Hennell of our Medical Thoracic Group. One appeared in the *Archives of Internal Medicine* for April, 1939, and the other in the *Journal of the Mount Sinai Hospital* for September, 1946.

Patients suffering from chronic pneumothorax naturally come to our medical men first. Since the latter traditionally are more conservative than we of the surgical guild, they have treated such cases almost routinely by the use of pleural irritants. They employ a saturated glucose solution of approximately 67 per cent concentration; and, when the lung is approximately three-quarters expanded, they introduce 10 to 15 c.c. of Lipiodol. They have treated 26 such cases up to the present, and, thus far, have been so successful that we of the Surgical Group have not been called upon to render surgical treatment. Perhaps we will some time in the future but, up to the present, we have not.

Of the 26 patients treated conservatively by our Medical Group, 23 have been cured with one course of injections. A course consists of four to six injections. Two of the 26 were cured with two courses and follow-up study could not be done in one. In other words, of the 25 patients in whom follow-up has been done, all are well.

Two patients with large air cysts of the lung were treated by the same method. The surgeons among us were shocked to see the medical men treat cysts of the lung in this way, but much to our amazement, the 2 patients recovered.

(*Slides.*) With regard to the first case that Dr. Brewer showed, you may recall that after the patient had been treated by underwater drainage, the lung expanded well. I think that our medical men would treat such a case conservatively. (*Slide.*) This is the film of a patient who already has had 67 per cent glucose introduced into the pleura. You will note that the lower portion of the lung has re-expanded. There is a large pneumothorax above. The heart is markedly displaced toward the right. (*Slide.*) Here is the film of the same patient after the introduction of Lipiodol. I might add that when irritating substances are introduced into the pleura, the patient is instructed to lie on his side for approximately twenty-four hours with the well side down so that the solution may come in contact with the surface of the lung. (*Slide.*) Here is a film of the same patient subsequently, with the lung fully expanded. Here you see globules of Lipiodol in the pleura, most of it lying behind the heart. (*Slide.*)

Here is a final case in which you see the lung completely collapsed. (*Slide.*) This is the last film demonstrating complete re-expansion of the lung and scattered droplets of Lipiodol in the pleura.

DR. FRANCIS WOODS, Brookline, Mass.—I would like to mention a small group of cases we have seen that falls, I think, into the same category. These patients have had repeated spontaneous pneumothorax, as much as six and sometimes eight times, and I think in these cases we have justifiably done explorations on the chests and found a small cyst which was the source of the leak, ligated it or removed it by segmental resection. In one or two who had repeated spontaneous pneumothorax no leak was found. Therefore, I would like to call attention to a group who do not have chronic spontaneous pneumothorax, but recurrent pneumothorax that I think should be treated in the same group.

DR. FELIX A. HUGHES, Jr., Memphis.—I think the plan of treatment presented by Dr. Brewer is rational and effective. The morbidity is much less in these patients if an aggressive attitude is taken toward the spontaneous pneumothorax. I would like to mention that of 15 patients with an acute spontaneous pneumothorax, treated with immediate closed thoracotomy, 13 had immediate re-expansion; 2 failed to have expansion and open thoracotomy was done with conservative resection of the bleb or cyst. I think the rupture or disruption of the lung or bleb may heal more rapidly and more permanently with the lung expanded than with it collapsed.

I would also like to present one slide which shows the membrane Dr. Brewer spoke of; here you can see a chronic spontaneous pneumothorax with one of the blebs excised. You can see other blebs in place in the lung.

DR. SHELDON E. DOMM, Knoxville.—Dr. Brewer has mentioned the rarity of autopsy confirmation in these cases. We would like to present a case in which there were unusual post-mortem findings. (Slides.)

This is a 14-year-old white boy, first seen with what appeared to be a simple acute spontaneous pneumothorax on the right, which responded to conservative management, but recurred in one month. Again the right lung re-expanded and appeared normal. However, two months later x-ray examination showed shadows suggestive of bullous emphysema in the right lower lobe. About one month after this development the patient was readmitted to the hospital in his third episode of right-sided spontaneous pneumothorax. Conservative measures, including an indwelling cannula, failed to relieve the dyspnea; subcutaneous and mediastinal emphysema ensued and were not relieved by supraclavicular and suprasternal incisions. X-ray examination three days later, on the day of death, showed another spontaneous pneumothorax to have occurred on the left. The patient died about six months after the first episode of spontaneous pneumothorax. Autopsy showed both lungs to be almost completely replaced by cyst formation with fibrosis, with emphysematous blebs studding the entire pleural surfaces. The pancreas was about one-fourth normal size and showed marked alteration of the normal architecture, notably a diffuse increase in connective tissue. This picture, however, differed from the usual findings in fibrocystic disease of the pancreas in that the pulmonary changes were decidely atypical.

This case was particularly interesting to us because of its diagnostic and post-mortem features, and the question whether surgical intervention would have been justified.

DR. ALFRED GOLDMAN, Los Angeles.—I wish to bring out two points which have not been mentioned, that were encountered in operating on 4 patients with chronic spontaneous pneumothorax associated with large cystic dilations which one would probably classify as giant bullae. The first point is that on making the incision to open the chest the patient's condition is usually not good from the anesthetist's point of view. The anesthetist complains of some difficulty with the anesthesia; since the cyst usually appears in the wound as soon as the pleura is opened one should immediately puncture the cyst. When this is done the anesthetist usually will say that the patient's condition is improving and anesthesia proceeds more smoothly. The second point I wish to make is regarding the demonstration, in one of these patients, of the leak in the lung. Dr. Brewer mentioned that one must use saline solution; however, sometimes the point of rupture is not obvious and in one of these patients a two inch cyst was completely enclosed within an apical adhesion so that all one saw on opening the chest was the collapsed lung and adhesion. This was a large vascular veillike adhesion, and on re-expansion of the lung and making pressure with the bag by the anesthetist, one could see the balloonlike cyst structure inflating through the walls of the adhesion. On using saline solution and putting the patient in the Trendelenburg position, a small rupture was discovered at the base of the adhesion. Thus one should look for a cyst within an adhesion if adhesions are present and no other obvious source for the air leak is discovered.

DR. BRIAN B. BLADES, Washington.—Dr. Alexander has mentioned my interest in the procedure of poudrage. I want to make it clear that, in my opinion at least, poudrage has no place in treatment of chronic pneumothorax. It is useful, however, for the patient who has frequent bouts of spontaneous pneumothorax of nontuberculous origin, and in whom at operation many small blebs are found over the surfaces of the lung. In some cases there may be dozens of blebs, all of which are the potential source of an air leak. I feel very strongly that if the seriousness of pneumothorax justifies operation, it justifies open operation, enabling direct inspection of the lung. In many cases a single, two, or perhaps three blebs are seen which can be excised. The use of poudrage is rather limited and only applicable to those cases in which there are many small blebs.

DR. BREWER.—I wish to thank all the discussants for the great interest that they have shown in this discussion. I think Dr. Touroff has demonstrated very well the effect of pleural irritants. Certainly, the second case that he presented showed considerable pleural

thickening. I think the use of irritants may be justified very early in the course of the disease, when the lung is mobile. Then, only, the injection of mild irritants might be tried. All who have been through the poudrage technique, with iodized talc, developed by Norman Bethune, as a preliminary procedure to lobectomy, know that the violent pleuritis frequently seen was serious; the patient often had more reaction to the poudrage than to the lobectomy that followed. This procedure, as you know, is not commonly used any more. Of course, if there are bilaterally destroyed lungs, no treatment will avail. However, the local resection (and by local resection we mean only the bleb that is responsible for the pneumothorax or a large thin bleb that is adjacent) will result in closing of the pleural aperture with minimal loss of pulmonary tissue. Certainly, we have reached the stage in the management of thoracic diseases where we must think of preserving pulmonary function and not merely the palliative sealing off of the pleural aperture.

We admit that in many instances the production of a violent pleuritis will seal over the pleural opening. However, it may take as long as one year for this sealing off to take place. This result is effected at the expense of pulmonary function.

SEGMENTAL RESECTION IN PULMONARY DISEASES

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INTRODUCTION

SEGMENTAL resection of the lung is an operation designed to rid the patient of the major diseased components with the minimum sacrifice of normal functioning parenchyma. This result is achieved if (1) the proper segment or segments are removed; (2) the residual circulation is not impaired; and (3) re-expansion is complete and immediate.

REMOVAL OF THE DISEASED SEGMENT

The wrong segment was removed in two instances in this series as demonstrated by postoperative bronchograms. Other mistakes may have been made in the few nontuberculous patients who did not respond to the request for follow-up bronchographic studies. The known errors, however, occurred early in our experience and if the present clinical evidence is reliable the number of other errors are few.

Basic anatomic knowledge of the bronchopulmonary segments is essential to a successful result, but often disease so distorts the normal anatomy that the surgeon is provided a new surgical experience at each resection. These alterations by disease in addition to the large number of (anticipated) anatomic variations have taught us the necessity for a standardized operative approach.

The first objective has been the identification and isolation of the bronchus serving the diseased segment. In many instances it is possible to divide the proper bronchus before ligation of *any* pulmonary vessel. In exposing the bronchus and its main divisions it may be necessary to define several branches of the pulmonary artery to a lobe before the proper bronchus may be selected.

The second objective is ligation of the arterial supply to the segment. It is often technically much easier to divide the artery before the bronchus, but the inconsistency of the vascular pattern in comparison with that of the bronchial tree has influenced the order of our approach. Once the bronchus is divided and gentle traction is put upon it, the proper segmental artery quickly joins it. Only then is it safe to ligate and divide it.

The final objective in the removal of the bronchopulmonary segment is ligation of the pulmonary veins. Suffice it to say, at this time, that it is a good general rule to ligate as few veins as possible at the hilar level.

The opinions or assertions expressed in this paper are private ones of the authors and are not to be construed to be official or reflecting the views of the Navy Department or the Naval Service at large.

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RESIDUAL CIRCULATION

The veins are intersegmental and receive tributaries from two adjacent segments. Ligation of the vein at the hilum may cause, thereby, a passive congestion of the segments to be left behind. Swan[7] has recently reported the pathologic changes associated with ligation of the pulmonary veins. These changes are mainly those of passive congestion, and if after segmental resection postoperative hemoptysis or streaking occurs beyond the first few days, it is suggestive evidence that venous obstruction is present in the residual circulation.

The proper segmental plane for dissection is disclosed as soon as gentle traction is applied upon the divided segmental bronchus and artery. It is in this plane that the small venous tributaries empty into the main intersegmental vein. As the dissection progresses the larger tributaries may be clamped, but the smaller ones are usually torn across unseen at their junction with the intersegmental vein. A small amount of bleeding occurs, as a result, but this soon stops, and the intersegmental vein is easily visible on the raw surface of the residual segments. Preservation of this vein is an important refinement in the technique of segmental resection. This procedure, as outlined, is a true segmental resection.

RE-EXPANSION

Complete and immediate re-expansion of the lobe and segments is essential to a successful result because (1) infection may be prevented if the dead space is obliterated and (2) maximum function of the residual parenchyma is obtained.

Obliteration of all dead space is a basic principle in surgery if infection is to be avoided. It is an accepted fact that the pleural cavity is always contaminated in the resection of the lung, a lobe, or any of its segments, but it is also recognized that the pleura like the peritoneum is not entirely defenseless. Therefore, if one is careful to reduce the contamination to a minimum and is insistent upon complete and immediate re-expansion, an empyema is not likely to occur. This principle was true and effective before the use of chemotherapy, yet even with chemotherapy an empyema occasionally develops if this fundamental principle is violated.

Maximum respiratory function, of course, is the main objective in establishing complete and immediate re-expansion, but if this end is to be realized, constant and controlled intrapleural suction is considered essential. A few small fistulas usually are present after a segmental resection and in a sense the problem is similar to the one which occurs in a spontaneous pneumothorax. Its treatment, however, is quite different. The lung or segments should not be allowed to recoil until the fistulas have closed, because a serosanguineous effusion (simulating a hemothorax) accompanies the thoracotomy, and the lung may be trapped in the partially collapsed position. Continuous but controlled suction which can be adjusted to maintain the normal intrapleural pressure seems preferable and is, of course, more physiologic. Such suction removes air faster than it leaks through the fistulas and in this way a normal negative pressure is established in the pleural cavity.

Adjacent structures soon fuse with the raw area and seal the fistulas so that re-expansion is rapid and complete. For example, if the superior segment of the lower lobe has been preserved, the diaphragm soon rises to seal the raw surface; if the apical posterior segment of the left upper lobe has been removed, the raw area of the anterior segment is soon fused in the vertebral gutter; and in a similar way, in each segment removed, the diaphragm, the adjacent lobe, the chest wall, or the mediastinum soon fuse with the raw area and all fistulas are closed.

The actual contribution of the residual segment to total lung function is probably not great, but as a "filler" it contributes greatly to respiratory function. For example, in those cases in which the superior segment of the left lower lobe has been preserved, a large pyramidal space is filled by the segment. The diaphragm, as a result, not only retains its normal contour, but, more important, retains its pistonlike activity. On the other hand, if the entire lobe is removed the upper lobe must overdistend considerably to fill this space, and the diaphragm becomes elevated, flat, and fixed in the vertebral gutter. For obvious reasons, diaphragmatic fixation and overdistention of the lung are complications to be avoided whenever possible. Therefore, the main contribution of the residual segment is its influence upon the function of the diaphragm or the other components of the lung.

TECHNIQUE

The segmental resection technique which we have used was based upon the concepts of Churchill,[3] Blades,[1] Clagett,[4] and Overholt.[6] We began as did Churchill by identifying, isolating, and dividing the cardinal structures (except the vein) at the hilar level. We applied this technique to individual segments as suggested by Blades. We used Clagett's "traction-dissection" technique, but without closing the raw area as advocated by Overholt.

However, early in our experience we discovered the unreliable nature of the venous pattern and errors were made by ligation of the wrong veins. This led to a state of insecurity on our part, the solution to which was procrastination. We decided to put off the ligation of any vein as long as possible. Therefore, after selecting the proper bronchus and artery, the intersegmental plane was entered and the dissection continued until no further progress could be made without ligation of a vein. Once the segment was removed, however, there could be seen a large vein on the raw surface of the adjacent segment which bled at many points along its course. Only then did we understand the teachings of Boyden and Scannell,[2] that there were intersegmental veins which received blood from two adjacent segments. Finally, we realized that during the dissection we were tearing across venous tributaries which were enroute to join the main intersegmental vein. Overholt referred to them as "vessels" which were often indistinguishable from the smaller bronchioles and he recommended severing them. If bleeding occurred they were ligated, if not, they were traced as small bronchi to their termination and removed with the segment. Though we have refined our techniques and now clamp and ligate the larger tributaries, we still find it difficult to avoid traumatizing the intersegmental vein in all cases. The

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injury is often due to the fact that the inflammatory reaction, at one time, has extended across into the adjacent segment; the residuals of this process interfere with dissection along the intersegmental plane. Obviously, therefore, attempts at segmental resection during an acute inflammatory phase are to be avoided. In this connection, however, if during dissection the intersegmental plane seems to be obliterated by the chronic inflammatory reaction it may be advisable to return to the parent bronchus and select an adjacent bronchus which also is included in the resection. The entire inflammatory process is circumvented in this way. Occasionally, a subsegment of the adjacent segment will suffice. In other words, a segment and subsegmental resection are done.

Other techniques are indicated in some cases. For example, when the lesion is small the subsegment may be removed by local excision. A modification of this technique is the excision between clamps when the lesion is located along a fissure or the free edge of a lobe. Occasionally, it is preferable to begin the resection at the periphery. We have referred to this as the "outside-in" technique. Cysts, atelectatic segments, or even chronic lung abscesses may be treated in this way. Our experience would lead us to conclude, however, that those methods which leave the raw area exposed and do not alter the contour of the residual lobes for the reasons we have pointed out are preferable to those which prevent the lung from "breathing" by approximation of the pleural surfaces.

SUMMARY OF CLINICAL MATERIAL*

The conclusions on segmental resection are based on the experience gained in this series of 100 segmental resections. A total of 91 operations were performed on 85 patients in the last two years (Table I). This type of operation has been done on 37 patients with bronchiectasis, 30 with tuberculosis, 13 with lung abscess, 2 with cysts of the lung, and 1 case each of bronchiogenic carcinoma, bronchial adenoma, and coccidiomycosis.

TABLE I. RÉSUMÉ OF THE STATISTICS

	PATIENTS	OPERATIONS	SEGMENTS RESECTED	COMPLICATIONS	DEATHS
Bronchiectasis	37	43	52	19	1
Tuberculosis	30	30	30	10	1
Lung abscess	13	13	13	1	1
Other diseases:					
(1) Cysts	2	2	2	0	0
(2) Coccidiomycosis	1	1	1	0	0
(3) Bronchial adenoma	1	1	1	0	0
(4) Bronchiogenic carcinoma	1	1	1	0	0
Total	85	91	100	30	3

BRONCHIECTASIS

In the cases of bronchiectasis (Table II) there were a total of nineteen complications, but several of these complications occurred in the same patient so that the complications occurred in less than the 44 per cent which seems to

*The material for this paper came from the Chest Surgical Service of Bellevue, St. Albans, Halloran, and Triboro Hospitals. In eight of the cases from the Service at Bellevue in which this technique was used, the patients were not operated upon by the authors, but permission for their inclusion was granted by Dr. Berry, the Chief of the Thoracic Service at Bellevue.

TABLE II. SEGMENTAL RESECTION FOR BRONCHIECTASIS

PATIENTS	OPERATIONS	SEGMENTS RESECTED	COMPLICATIONS	DEATHS
37	43	52	19	1
Complications in the Bronchiectasis Cases				
	Spread, contralateral		1	(Patchy pneumonia)
	Bronchopleural fistula		3	
	Empyema without bronchopleural fistula		1	
	Incomplete or delayed re-expansion		5	
	Atelectasis		3	
	Recurrent spontaneous pneumothorax		3	
	Pulmonary embolus		1	

be indicated. The complications were not serious and added little to the dangers of the operation in most instances. Atelectasis required bronchoscopy in only one instance, while the other two patients responded rapidly to intratracheal aspirations with a catheter. The incomplete or delayed re-expansions required suction on intercostal tubes longer than the forty-eight to seventy-two hour period usually necessary. One patient required repeated aspirations over a period of several months. The cases of spontaneous (recurrent) pneumothorax required closed thoracotomy drainage by an intercostal tube connected to suction for only the forty-eight to seventy-two hour period. The three patients with bronchopleural fistula who developed localized empyema required intercostal tube drainage for a period of several weeks. The one case of empyema without bronchopleural fistula probably was due to breaking into a localized abscess surrounding a piece of shrapnel in the lung which was believed to be the cause of the bronchiectasis and was removed at the operation. The one mortality in this group was probably due to pulmonary embolus.

TUBERCULOSIS

In the thirty cases where segmental resection was done for tuberculosis (Table III), the early postoperative complications were not as severe nor as numerous as would be anticipated. There were three of these cases in which a lobectomy was performed on the same side at the same operation and one case in which segments were removed from two lobes at the same operation, so the complications must be considered in light of these compound procedures.

TABLE III. SEGMENTAL RESECTION FOR TUBERCULOSIS

PATIENTS	OPERATIONS	SEGMENTS RESECTED	COMPLICATIONS	DEATHS
30	30	30	10	1
Complications in the Tuberculosis Cases				
	Spreads, contralateral (or reactivation)		1	
	Spreads, homolateral (or reactivation)		2	
	Bronchopleural fistula		3	
	Empyema without bronchopleural fistula		0	
	Incomplete or delayed re-expansion		2	
	Atelectasis		1	
	Recurrent spontaneous pneumothorax		0	
	Coronary occlusion		1	
	Wound infections		0	

The spread of the tuberculous infection is a danger in any operation for tuberculosis, yet we have noted only one case of contralateral spread and this was an increased activity of a previously noted disease probably because of a bronchopleural fistula, and the area of reactivation improved when re-expansion was obtained by re-establishing suction through an intercostal tube. The incomplete or delayed re-expansion of the remaining segments of the lung we believe is due to fibrosis from the healed processes in the remaining lung and is an indication for a relaxing type of thoracoplasty. The one death thought to be due to coronary insufficiency is probably a mortality of surgery in general rather than to the type of procedure performed.

The final evaluation of these cases of segmental resection for tubreculosis will require many years. All that can be said at this time is that the results thus far are such that we expect to continue using the procedure in carefully selected cases.

LUNG ABSCESS

Segmental resection for lung abscess (Table IV) is done only in the chronic forms of the disease or subsequent to drainage. However, resection was done in one case of acute putrid lung abscess. Pulmonary gangrene of the residual lobe occurred followed by a severe hemoptysis and death. At the present time, we believe that segmental resection should not be performed in cases of acute putrid lung abscess. The remaining twelve cases with chronic lung abscess were without complications. There were two cases in which resection was done with the tube still in place following drainage.

TABLE IV. SEGMENTAL RESECTION FOR LUNG ABSCESS

PATIENTS	OPERATIONS	SEGMENTS RESECTED	COMPLICATIONS	DEATHS
13	13	13	1	1
Complications in the Lung Abscess Cases				
	Pulmonary gangrene		1	
	Bronchopleural fistula		0	
	Empyema		0	

In a summary of the statistics there were 85 patients reported who had a total of 100 segmental resections requiring 91 operations with 30 complications reported, most of which could be considered minor. There were 3 deaths, or a mortality of 3.3 per cent, but only 1 of these deaths should be attributed to the type of operation performed.

DISCUSSION

Identification and division of the bronchus is mainly done to control secretions. By persistent effort and experience it is possible to become more and more adapt at this maneuver. Certainly the effort should be made in each case, and if ligation of a branch of the pulmonary artery facilitates early isolation of the bronchus, it should be done—especially in patients with tuberculosis. Identification of the bronchus is a matter of meticulous dissection. Prior to the operation, of course, roentgenograms, bronchograms, bronchoscopy, and laminag-

raphy allow the surgeon a preview of the task at hand. Since bronchograms are undesirable studies in patients with tuberculosis we have followed Klopstock's recommendation of using not only the posteroanterior, but lateral laminagraphic studies as well. The information gained by laminagraphy is actually more reliable than the surgeons' palpation. On occasion, at the operation we have changed some of our preoperative decisions and performed a lobectomy rather than a segmental resection. When this has occurred, postoperative examination of the surgical specimen more frequently coincides with the laminagraphic studies than with the surgeons' impression from palpation.

The *residual circulation* is significant in relation to the definition of segmental resection. Segmental resection, as generally used, is a term with an ambiguous meaning. In the past, and in this series, segmental resection has included the removal of the lingula, the superior segment of the lower lobe, and all the various segments of the other lobes. Nelson, Churchill, and Blades pointed out some time ago that the lingula and superior segment were essentially individual lobes and were occasionally separated from the other lobes by partial or nearly complete fissures. Blades more exactly reserved the term segmental resection for the removal of parts of a lobe. In principle, the technical removal of the lingula is quite different from removing one of its segments. For example, when the lingula is to be removed the cardinal structures including the vein are ligated at the hilum. On the contrary, if one of the segments of the lingula is to be removed the vein is not ligated at the hilum, because its main tributary is the intersegmental vein which receives blood from both segments. The latter procedure is more exactly called a segmental resection.

Obliteration of the pleural dead space by complete and immediate re-expansion is the most important single factor in the postoperative management. In those cases where good re-expansion was obtained the complications were few. The problems of re-expansion, assuming the bronchi are patent, depend upon, mainly: (1) the degree of pre-existing emphysema, (2) the duration of the disease, and (3) the amount of parenchyema to be removed.

Pre-existing emphysema, regardless of the etiological factor, is undesirable because such lungs are inelastic and already may be occupying a space closely approaching its maximum distensibility. Segmental resection rather than a lobectomy is particularly useful in these patients because the demand on the already enlarged lobes to fill a still larger space is, by comparison, a minor one.

The *duration* of the disease is significant in that the time factor influences greatly the compensatory factors at work. As the disease heals and contracts, the other segments often enlarge, especially if the disease occurred early in the patient's life. Removal, therefore, of the diseased segments imposes a minor demand upon the residual segments in obliterating the pleural dead space. The best results with the fewest complications may be anticipated in patients whose disease has been in existence since childhood.

The *amount* of lung removed greatly influences morbidity figures. If an upper lobe and superior segment of the lower lobe are removed, the demand upon the basilar segments to overdistend and fill the hemithorax is a major one. Add to this the hypothetical existence of extensive emphysema occurring late

in adult life when the compensatory factors are less effective and a most hazardous situation is created for the development of postoperative complications. In such cases it is often better to perform a thoracoplasty first, which tailors the thorax to fit residual segments.

The immediate morbidity rate of segmental resection is slightly greater than that of lobectomy. The operation takes longer to perform, demands a more meticulous dissection, and requires greater postoperative vigilance. But the late morbidity figures may prove to justify it because not only is the disease removed, but maximum pulmonary function is preserved.

CONCLUSIONS

1. The term segmental resection exactly used applies only to the removal of the bronchovascular components which combine to form a lobe.

2. The lingula and superior segment of the lower lobes are essentially individual lobes whose "primitive" fissure is usually undeveloped. The lingula is composed of two segments and the superior segment of the lower lobe is composed of three.

3. Preservation of the intersegmental vein in performing a segmental resection is a worthy refinement in technique.

4. Morbidity factors are reduced to the minimum by obliteration of the dead space through the complete and immediate re-expansion of the lung.

5. Continuous but controlled intrapleural suction lends the greatest assurance to the maintenance of normal physiologic re-expansion.

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(See discussion on page 220.)

SEGMENTAL PULMONARY RESECTION

Details of Technique and Results

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SINCE segmental resection of the lung was conceived by Churchill and Belsey,[2] this operative procedure has undergone neglect in childhood, enthusiasm and doubt in adolescence; it is now ready for acceptance of its maturity.

The development of segmental pulmonary resection is the result of the contributions of a succession of individuals. One of the first was the report of Nelson[1] who described each lung as having four basic parts, each of which was a separate anatomic unit. Later Alfred Goldman[3] drew attention to the fact that a lung macerated in weakly corrosive solution tended to separate into segments. Churchill and Belsey applied the increasing anatomic knowledge to pulmonary resection and proposed and carried out segmental resections. Later contributions were made by Kent and Blades[4] and Blades.[5] Clagett[6] reported a technique of lingulectomy wherein the intersegmental resection was commenced in the hilum and clamps were not used. He oversewed the pleural margins with interrupted sutures. Overholt and his associates[7-11] developed a technique of segmental resection and applied it to all segments and some subsegments. This technique was featured by blunt dissection of the intersegmental plane commencing in the hilum, avoidance of the use of clamps, and leaving the exposed intersegmental surface free to expand. Boyden[12] emphasized the intersegmental arrangement of the veins and many surgeons were aware of their presence in the intersegmental plane at the time of segmental resection. Betts[13] pointed out this arrangement to Ramsay.[14] The latter, following laboratory dissections, recommended that these veins be located in the hilum of the segment to be removed and followed as a guide to the intersegmental plane.

In its early development the procedure was largely but not entirely limited to lingulectomy by the clamp method. Abandonment of the clamp method and the persistent application of the individual ligation principle to all pulmonary segments by Overholt and his co-workers[7-11] featured the adolescent period. The discouragements at that time were principally the high incidence of complications; empyema occurred in 18 per cent[7] and bronchopleural fistula in 6 per cent.[7] These were the results of difficulties in the dissection of the intersegmental plane. They have been largely overcome by utilizing the intersegmental veins as guides to the plane following the suggestions of Ramsay.[14] With continued experience both bronchopleural fistula and empyema are uncommon and unexpected.

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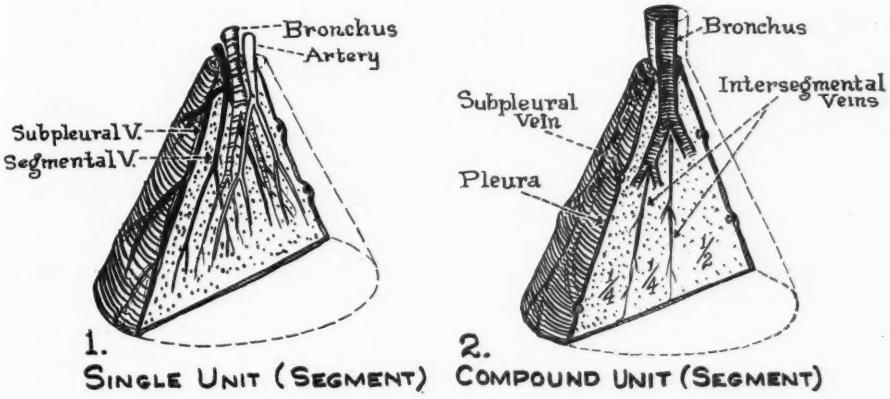
The purpose of this presentation is to record the details of operative technique as used today and the immediate postoperative results as found in the last twenty-eight consecutive segmental resections.

The steps of segmental pulmonary resection are essentially four in number. (1) The segmental artery is dissected out, ligated, and divided. (2) The segmental bronchus is cleared, ligated distally, divided, and the proximal stump closed. (3) The segmental vein, when present, is ligated and divided. (4) The segment to be removed is then separated from the lobe by locating the intersegmental vein in the hilum of the segment, and following this as a guide to the intersegmental plane. The first three steps are carried out in the order which is most convenient. The bronchus is located by palpation, the artery being adjacent. Care is taken to determine that they enter the segment to be removed. If subpleural veins, draining the diseased segment, are encountered, they are ligated and divided. Such veins are constantly found beneath the pleura of the lingula, the anterior segments of the upper lobes, and the middle lobe.

The arrangement of pulmonary veins can best be illustrated by considering the structure of the pulmonary unit or segment (Fig. 1). This we conceive is conical, covered by pleura, and contains an axially situated bronchus and artery. The principal veins lie on the surface and converge on the apex or hilum. There may be a small vein accompanying the artery and bronchus. When two or more of these units combine to form compound units or larger segments (Fig. 2) the surfaces in contact lose their pleural covering, and the subjacent veins become intersegmental in position. Elsewhere they remain as subpleural veins. Just as the bronchus and artery to a unit or segment are called segmental, so too is the vein which accompanies them. Thus, we have two types of veins: one central, accompanying the artery and bronchus and the segmental vein, and one peripheral; such a vein is either subpleural or intersegmental.

Finding the Intersegmental Vein.—The intersegmental vein is always to be found at the hilum of the segment in question and should be identified there rather than in the intersegmental plane. Distally there is danger that branching may have occurred and a branch followed between subsidiary segments. Just a thin covering of areolar tissue often keeps the intersegmental vein from view. Gentle dissection, either with scissors or a gauze pledget will readily expose it. Too much tension on the segment being removed will attenuate the vein, render it bloodless and white, and disguise it from recognition. The intersegmental vein usually bears the same anatomic relationship to the segmental artery and bronchus as does the principal lobar vein to the lobar artery and bronchus. Thus, in the upper lobe the intersegmental veins are generally anterior to the segmental arteries and bronchi, in the middle lobe anterior and inferior, and in the lower lobe posterior. Occasionally the intersegmental vein will receive the segmental vein as a tributary. A vein (Fig. 3), exposed in the hilum, may be a subpleural vein, a segmental vein, or an intersegmental vein leading between other segments than the ones to be separated. A subpleural vein can be seen to lie subpleurally on the surface of the lung. A segmental vein, when followed for a short distance distally, can be seen to accompany a bronchus. An intersegmental vein does not accompany an artery or bronchus. If it is the one

leading to the intersegmental plane to be dissected, it can be seen to run toward the junction between the diseased and the normal segments. This junction can be estimated from the configuration of the lung but is best demarcated by differential expansion of the diseased and healthy segments. Where the segment being removed adjoins more than one other segment, there usually will be a separate intersegmental vein for each separate surface (Fig. 4). Thus the posterior segment of the right upper lobe has an intersegmental vein and surface between it and the anterior segment and also between it and the apical segment. These two intersegmental surfaces form a V-shaped trough into which the posterior segment fits (Fig. 4).



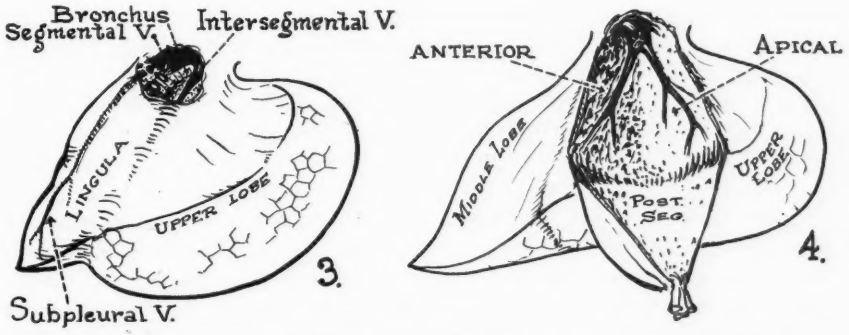


Fig. 1.—Our conception of the structural arrangement of the pulmonary unit. Note the central bronchus, artery, and segmental vein and the peripheral subpleural vein.

Fig. 2.—When such units are combined to form compound units or segments, some of the subpleural veins become intersegmental. The surgical segment illustrated here is shown divided into two quarter segments and a half segment corresponding to bronchial branching. Intersegmental veins demarcate the planes of separation.

Fig. 3.—A dissection of the hilum of the lingula: a subpleural vein is noted on the surface, a segmental vein accompanies a bronchus, and an intersegmental vein is shown between the lingula and the remainder of the upper lobe. Note that an intersegmental vein does not accompany an artery or bronchus.

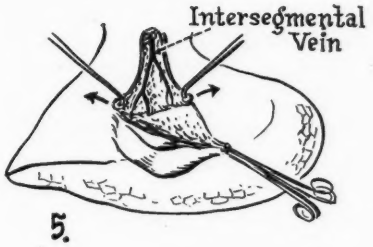
Fig. 4.—Segmental resection of the posterior segment of the right upper lobe is almost completed. Note the two intersegmental veins remaining on the freshly dissected surfaces of the anterior and apical segments.

After occlusion of the bronchus to the diseased portion, positive pressure is used to inflate the remaining lung, and then the contrast between the distended and collapsed parts outlines the approximate intersegmental plane. Frequently it is found that the diseased segment will fill with air although its bronchus has been occluded; in this situation deflation of the lung often will result in trapping of air within the diseased portion and provide the contrast in reverse.

Separating the Surfaces.—In carrying out the separation of the diseased tissue, exposure and gentleness are paramount. Exposure is best obtained by irrigating the surface free from blood and by keeping it flattened out. If the edges are allowed to roll inward, an accurate dissection is almost impossible. The margins of the segment can be gently retracted by divergent pressure at two opposite points (Fig. 5). Inflation of the lung will flatten out the intervening surface. Once the plane of dissection has been partially developed, counterpressure from the pleural surface at the level of dissection is of distinct aid in supporting the tissues during the process of separation (Fig. 6). The dissecting motion should be a side-to-side sweep between the intersegmental vein and its branches on the healthy side and the diseased tissue on the other (Fig. 7). The surfaces separate easily unless there is considerable fibrosis. The pressure is directed parallel to the plane rather than at right angles to it and may be accomplished with the rounded end of small curved scissors or the finger tip. Small venous tributaries, arising in the diseased segment, and any fibrous strands are cut as encountered (Fig. 7). It has been determined by one of us (B. H. R.) that in the normal fresh autopsy lung it is a simple and speedy matter to insert two index fingers along the central axis of the intersegmental vein, to move these fingers divergently, and to separate cleanly the two adjacent segments (Fig. 8). This maneuver is successful also in clinical cases where the tissues of the intersegmental plane are normal. If any resistance is felt it is important to visualize and divide the cause which will be a fibrous band or a venous branch. Otherwise the finger may be deviated into the normal segment with resultant air leak. This was the reason for the single grade 2 fistula which developed in the present series. If there is considerable fibrosis in the intersegmental plane sharp dissection is necessary, all fibrous bands being precisely divided.

Sources of Error.—At the apex of the segmental cone the borders of the intersegmental plane are close together and the pleura here tends to resist the opening up of the plane. There is a temptation to start the dissection by incising the pleura and underlying parenchyma on each side. Division of the pleura alone may be of distinct help. Cutting the parenchyma is dangerous since bordering segments may be distorted by traction or by compensatory change, or may normally extend unexpectedly far down upon the side of the segment being removed. This is the case with the superior segment of the lower lobe which fits like a hood on the basal group with its posterior portion extending inferiorly over the basal segments.

Damage to the lung surface is usually the result of "digging in" with the dissecting instrument or pulling on venous branches (Fig. 9). "Digging in" is the consequence of directing pressure against the normal segmental surface



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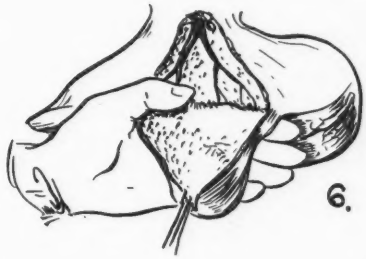

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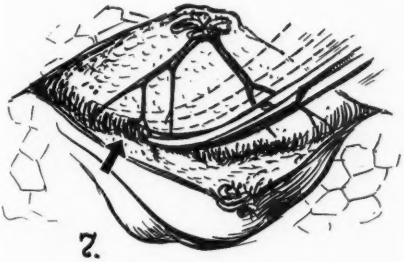

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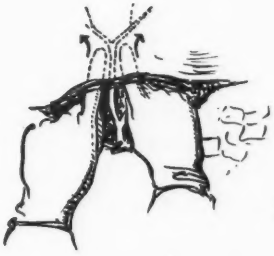

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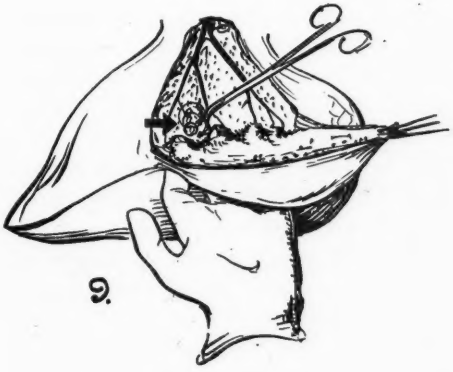

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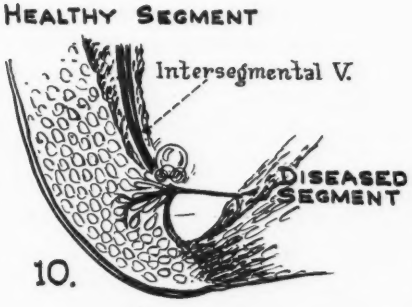

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Fig. 5.—Dissection of the intersegmental plane is facilitated by flattening the plane out by divergent retraction of the edges and inflation of the lung.

Fig. 6.—As the plane is developed, counterpressure from the pleural surface aids dissection.

Fig. 7.—This illustrates the side-to-side dissecting motion and division of a venous branch.

Fig. 8.—If the tissues of the intersegmental plane are normal, segments can be separated cleanly by divergent motion of the fingers inserted along the intersegmental vein.

Fig. 9.—Alveolar damage and air leak are illustrated. An incorrect type of counterpressure is shown. Note the flat side of the hand in Fig. 6.

Fig. 10.—Undue traction on venous tributaries may be transmitted to the normal segment with resultant parenchymal tears.

and is more likely to occur if the finger tips are used for counterpressure. The areas of lessened resistance between the points of finger pressure allow the dissector to furrow the normal tissue. Counterpressure should always be produced by a flat surface (Fig. 6). Traction or pulling on the venous branches is transmitted to the tributaries entering from the normal segment; these are anchored in alveolar tissue which is torn and air leak results (Fig. 10). It is advisable to cut the small venous branches as they are exposed. Only the larger veins will require ligation.

The intersegmental vein between the superior segment and the basal group of segments of a lower lobe corresponds to ramus V^6b of Boyden.[12] It has two principal branches, one anterior and one posterior. Dissection along the posterior branch should not be done on its posterior aspect since this may cause damage to the overlapping portion of the superior segment. Its bronchus of supply, the posterior ramus of the superior segmental bronchus (ramus B^6c of Boyden), may be exposed (Fig. 11).

As the pleural surface is approached, there is again a temptation to cut through the remaining tissue. It is better to follow the veins to the pleura where they will be seen to arch over the surfaces of both segments (Fig. 12). Occasionally, if too much traction is used on the diseased segment, these veins may be stretched to a thin whiteness which makes them difficult to recognize. Relaxation of the traction will overcome this difficulty.

We know of no instance where a remaining portion of a lobe has undergone torsion following segmental resection. However, because of the theoretical possibility of such occurrence any adhesions or partial fusions of fissure lines between residual segments of one lobe and adjacent lung are left undisturbed (Fig. 13). Care is also taken at the time of closure of the chest that after the lung is inflated the relative position of the remaining lung is a normal one. As long as inflation is maintained throughout the convalescence no torsion should occur.

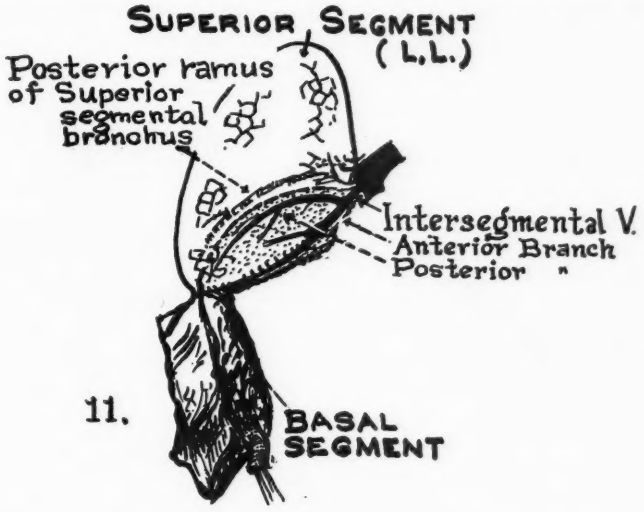
If the dissection is prolonged and continuous positive pressure is maintained, there may be a marked fall in blood pressure. In protracted dissections it is wise to interrupt the operation while the positive pressure is released.

Final Details.—While several dissections have been completed without a single air leak from the bare surface, there are usually one or two places which bubble slightly. If these are small and alveolar in origin they can be left safely to seal over in the first twenty-four hours. If the leak is more vigorous or if it is seen to be coming from an exposed bronchus, spontaneous closure cannot be depended upon. Attempts to suture these usually increase the amount of damage. Adequate control can be obtained by grasping the margins of the defect, with or without the adjacent parenchyma, between the teeth of fine forceps and by ligating the tissue so approximated (Fig. 14). All bleeding and significant air leaks having been controlled, nothing further is done to the surface.

To aid in re-expansion of the remaining lung all pleural attachments are separated. If the lower lobe or basal segments remain, the pulmonary ligament is divided. This permits the remaining lung immediately to rise higher within

the thorax, come in apposition with the pleural dome, and prevent apical accumulation of air or fluid. If full re-expansion of remaining segments is restrained by a pleural membrane, decortication should be practiced.

Prior to closure of the wound two catheters are placed intrapleurally. One is placed in the apex for the removal of air and brought out through the anterior portion of the incision. The other is placed just above the diaphragm for the



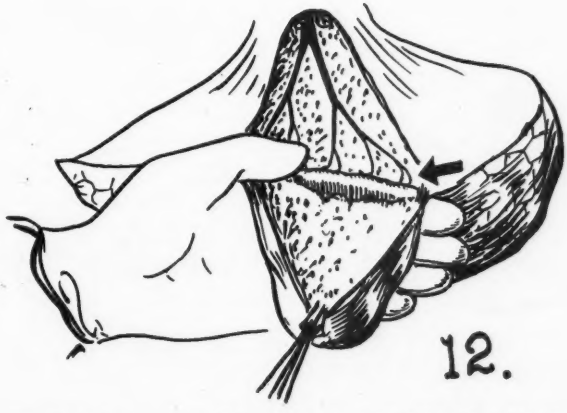


Fig. 11.—The posterior ramus of the superior segmental bronchus lies close to the intersegmental surface between the superior and basal segments of the lower lobe; it is easily injured.

Fig. 12.—The intersegmental veins are followed clear to the pleura before the latter is divided.

removal of fluid and is brought out through a small stab wound in the eleventh or tenth interspace. Care is taken that the catheter tip will not project into the bared segmental surface.

Postoperatively everything possible is done to obtain immediate and complete re-expansion of the remaining lung. To this end the catheters are connected to suction and water-trap drainage and the airways are kept clear. When the lung is completely re-expanded and no air leak is demonstrable, the intrapleural catheters are removed.

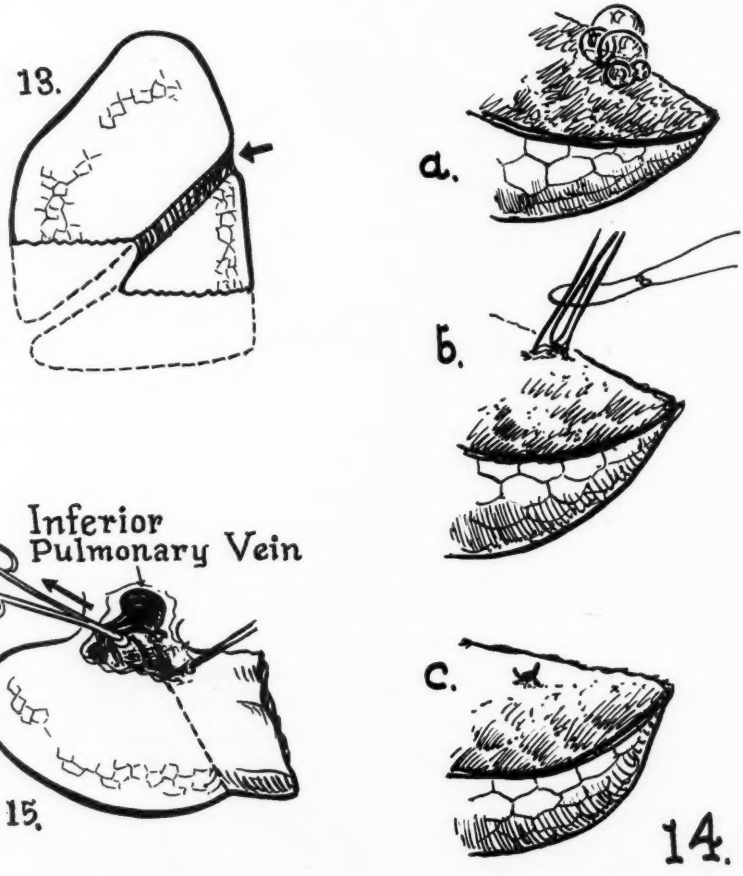


Fig. 13.—Adhesions between remaining parts of the lung are left intact to prevent torsion.

Fig. 14.—Control of significant air leaks is obtained by grasping and ligating the margins. Attempts to suture result in larger leaks.

Fig. 15.—Obstruction of the vein from the superior segment of a lower lobe may lead to engorgement of the segment. This may occur when exposing the basal bronchus for resection of the basal segments of a lower lobe.

RESULTS

All the segmental resections performed for any reason from September, 1948, to Feb. 10, 1949, have been reviewed.* Although our total experience in segmental resection now approximates 200 cases, there have been continual modifications in technique from that date until September, 1948. Since that month the details of the operation, as have been described, have been adhered to. Of the twenty-eight patients, nineteen were female and nine male. The average was just over 30 years of age, the youngest patient being 10 and the oldest 55.

TABLE I

DISEASE		NUMBER
Bronchiectasis		23
Unilateral	10	
Bilateral	13	
Tuberculosis		4
Cavitary disease	2	
Tuberculoma	2	
Chronic Abscess		1
Total		28

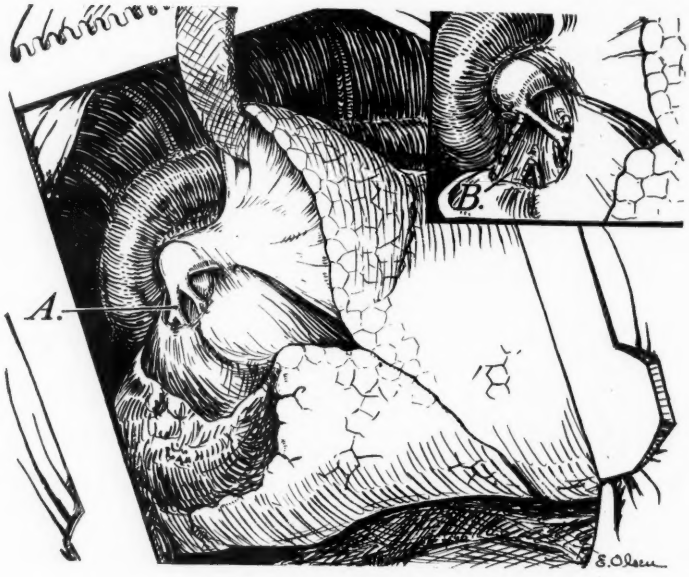
Table I shows the disease process in each case. Five of the patients with bronchiectasis had had resections on one side six or more months previously. There were two operations for cavitary tuberculosis where the superior segment of the lower lobe was removed with the upper lobe.

TABLE II. SEGMENTS REMOVED

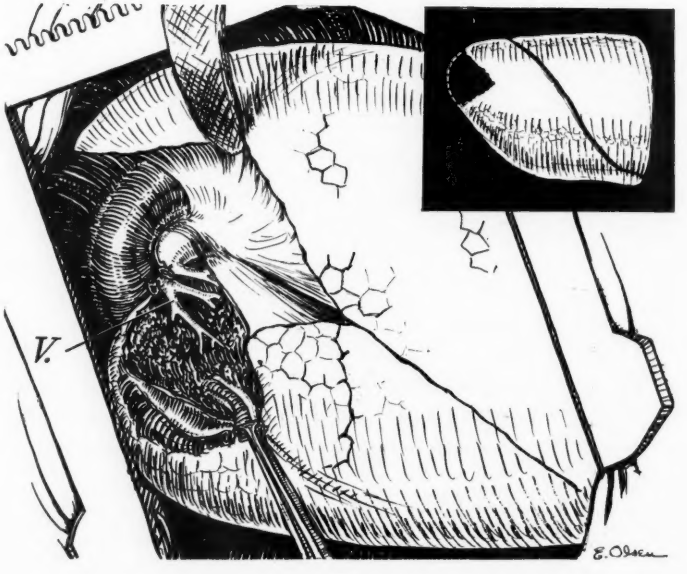
RIGHT LUNG		LEFT LUNG	
Upper lobe		Upper lobe	
Posterior	1	Lingula and anterior	1
Anterior	1	Inferior lingular	2
Anterior subsegment	1	Lingula complete	8
Middle lobe		Lower lobe	
Medial	1	Superior	1
Lower lobe		Subsegment of superior	1
Superior	3	Lateral basal	1
Medial basal	1	Basal group	11
Basal group	4		
Medial and posterior basal	1		

Table II lists the segments resected. Often these operations were combined with lobectomy of an upper, middle, or lower lobe as the distribution of the disease process indicated. Ten patients had two segments removed; eighteen had just one.

*Although these twenty-eight cases are all the segmental resections that were performed in this period for the definitive treatment of disease, several others were done as biopsies of undiagnosed lesions of the lung. This use of segmental resection is one of the most important of all, for more and more survey lesions are being discovered. If the patient has no symptoms and the lesion cannot be clinically diagnosed, some more conservative method than lobectomy or pneumonectomy must be offered. Early lesions often do not involve the lung surface and cannot be safely biopsied in the usual manner. We consider wedge resection nonanatomic and likely to produce air leak, infection, and spillage of tumor cells. Segmental resection affords an easily performed biopsy. If the lesion turns out to be benign, nothing further need be done. All the healthy pulmonary tissue has been conserved and the treatment for the isolated lesion has been ideal. Both the tuberculomas in this group were survey lesions managed according to this plan.



A.



B.

Fig. 16, *A*.—Resection of the apical segment of a left upper lobe. The artery (*A*) to the apical segment is exposed. The branch to the posterior segment is also seen.

Insert: The artery is divided. The bronchus (B) to the apical segment is exposed and the posterior segmental bronchus is also visible.

Fig. 16, *B*.—The bronchus is divided and dissection of the intersegmental plane is almost complete. Note the intersegmental veins (*V*).

Insert: Diagram showing left lung after resection of apical segment.

In fifteen cases the lung was completely re-expanded and both catheters were removed within the first twenty-four hours, in ten within forty-eight hours, and in the remaining two on the third and fifth days, respectively. This last patient had an upper lobectomy with segmental resection of the superior division of the lower lobe for tuberculosis. The residual lower lobe was not decorticated to prevent overexpansion; thoracoplasty was to follow. One patient died postoperatively.

The average postoperative hospitalization was 14.0 days. The shortest period was ten days and the longest nineteen days. The discharge date varied somewhat depending upon the distance to be traveled. Formerly the hospital stay averaged three weeks.

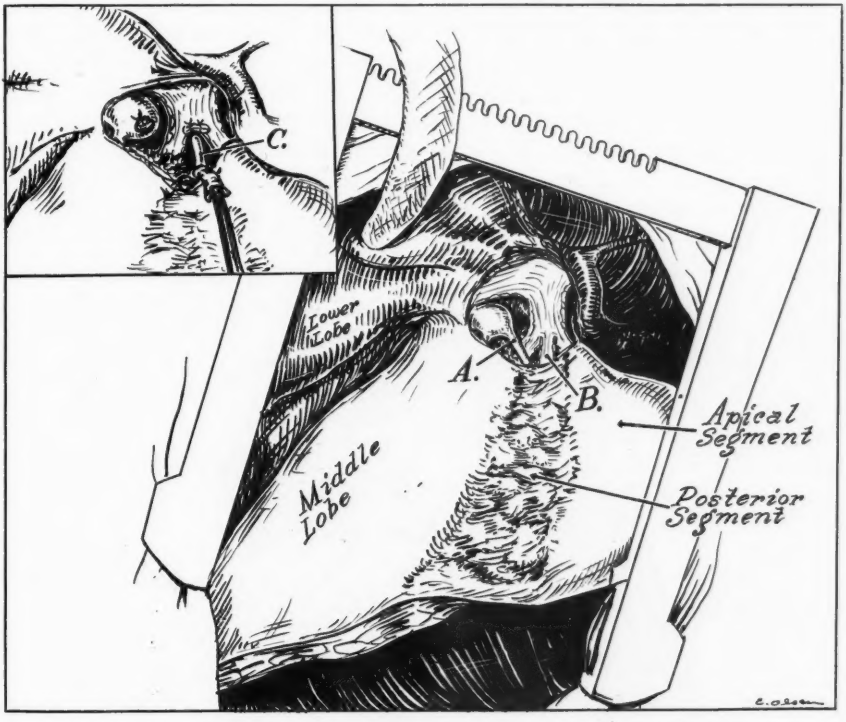


Fig. 17, *A*.—Resection of the posterior segment of a right upper lobe. An accessory artery to the posterior segment (*A*) is exposed and the bronchus (*B*) to the upper lobe and its three branches are exposed.

Insert: The bronchus and accessory artery to the posterior segment are divided. The posterior segmental artery (*C*) is now exposed lying anterior to the divided bronchus.

Table III lists the complications. There were no empyemas, two minor fistulas, and one death. Fistulas are graded first degree if the leak is so small that it is easily controlled by aspiration, second degree if it requires the reinsertion of an intrapleural catheter to obtain re-expansion, and third degree if it requires operative closure with or without decortication of the remaining lung tissue. The one second-degree fistula closed and the lung re-expanded

within twenty-four hours following reinsertion of an intrapleural catheter which was connected for suction. The one death occurred from pulmonary insufficiency aggravated by inability to raise the bronchial secretions.

TABLE III

COMPLICATIONS		
Empyema		0
Fistula		2
1st degree	1	
2nd degree	1	
3rd degree	0	
Death		1
Engorgement of superior segment		2
Total		5

This 24-year-old girl had had the middle lobe, basal group, and anterior segment of the upper lobe removed from the right lung on March 23, 1948. On October 11 the basal group and lingula were removed from the left lung. Postoperatively she was cyanotic in spite of re-expansion of the remaining lung on the left and no evidence of air leak from the intersegmental surfaces. Four

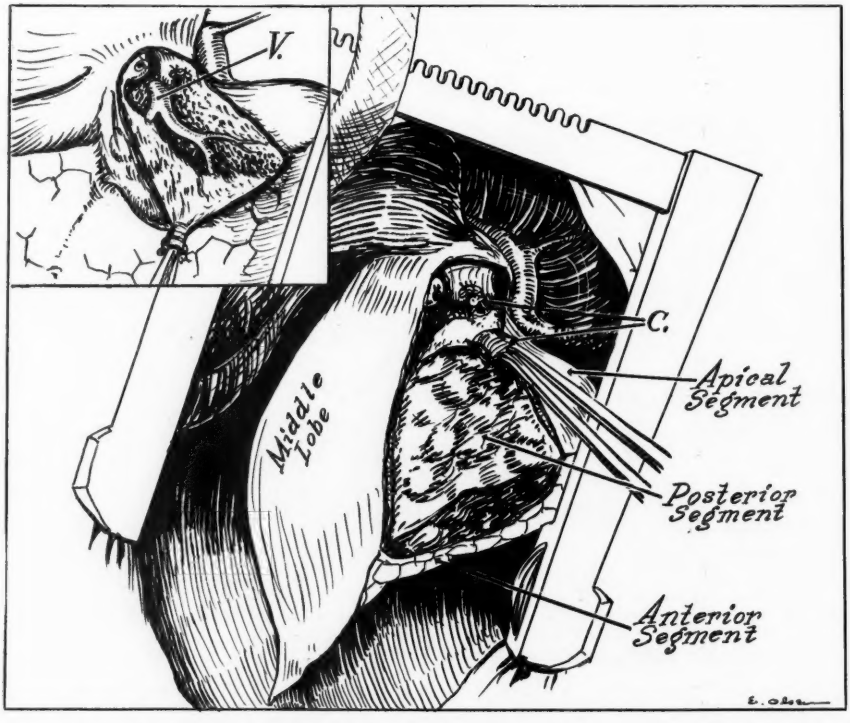


Fig. 17, *B*.—The posterior segmental artery has been divided. The intersegmental vein is obscured by areolar tissue. Note that the middle lobe and upper lobe are fused in this instance.

Insert: Dissection of the intersegmental plane between the posterior segment and the apical segment is completed. Dissection of the plane between the posterior segment and the anterior segment is just started. Note the intersegmental veins (*V*) remaining on the intersegmental surfaces.

hours postoperatively she had sudden difficulty in breathing. Although tracheal aspiration was successful in relieving her, she almost immediately had a second attack and succumbed before aspiration could be carried out. Autopsy revealed bronchiectasis in all but one of the remaining pulmonary segments and less than ½ c.c. of thin secretion in the left bronchial tree. The superior segment of the lower lobe was engorged with blood. The intersegmental surfaces were dry and no air leak could be demonstrated.

The engorgement of the remaining superior segment of the lower lobe following segmental resection of the basal group is a complication never recognized by us previously. This was not accompanied by necrosis. One other patient had a slight postoperative fever associated with an increase in density of the remaining superior segment as demonstrated by roentgenogram. These signs spontaneously disappeared in twelve days. We believe that this may represent a second case of engorgement. It is considered likely that this complication is produced by obstruction of the venous drainage from the superior segment. Retraction of this vein or torsion of the lobe, produced by attempts to obtain exposure of the basal bronchi, may obstruct it for a varying period

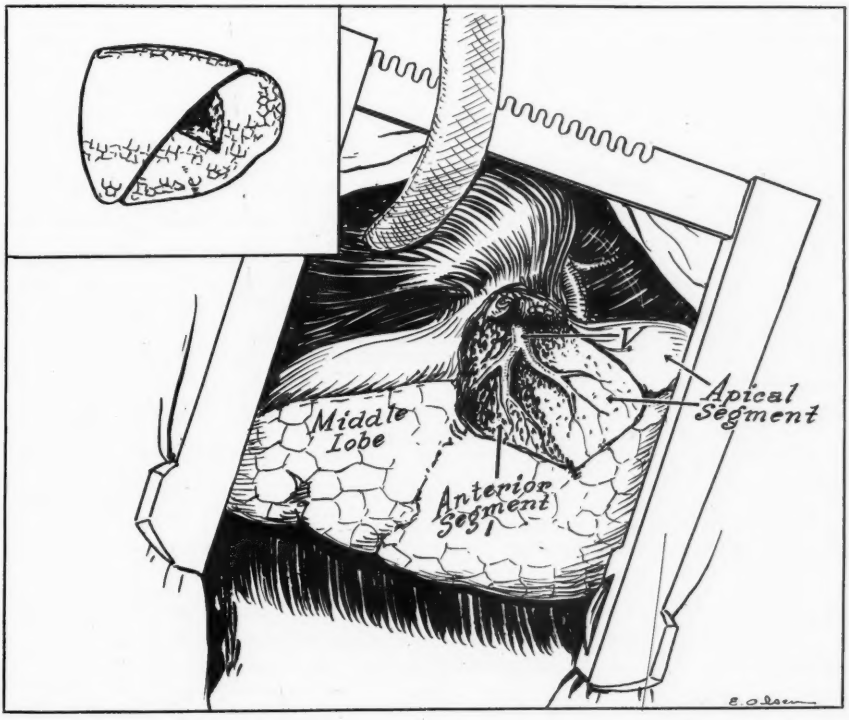


Fig. 17, *C*.—Posterior segment is completely removed, intersegmental veins (*V*) remaining on both the intersegmental surfaces.

Insert: Diagram showing right lung after resection of posterior segment.

of time (Fig. 15). Since the complication has been recognized we have carried out the mobilization of the basal bronchi from the anterior and inferior aspects as much as possible and have taken care not to obstruct the vein in question.

The value of segmental resection was emphasized by the anomalous condition found in a 10-year-old girl. She had bronchiectasis limited to the lingula and basal segments of the left lower lobe. At operation she was found to have a lung consisting of a single lobe without any trace of a fissure. It was not possible to expose the segmental arteries in the fissure as is customary, but by dividing the segmental bronchi and veins first, the arteries were dissected free without difficulty; then the intersegmental veins were located and followed as guides to the two intersegmental planes. No air leak came from the bare surfaces; the remaining lung re-expanded almost immediately and the intrapleural catheters were removed in less than twenty-four hours. She was discharged on the thirteenth postoperative day.

SUMMARY

The details of pulmonary segmental resection, with special attention to dissection of the intersegmental plane, have been described and illustrated. Emphasis is placed on using the intersegmental vein as a guide to the plane. There has been a marked reduction in complications.

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DISCUSSION ON "SEGMENTAL RESECTION IN PULMONARY DISEASES" BY DR. J. MAXWELL CHAMBERLAIN AND COMMANDER THOMAS C. RYAN,* AND "SEGMENTAL PULMONARY RESECTION" BY DR. RICHARD H. OVERHOLT, DR. FRANCIS M. WOODS, AND DR. BEATTY H. RAMSAY

DR. PAUL C. SAMSON, Oakland, Calif.—This is, of course, a very interesting experience to which we have listened. Dr. Dugan and I would like to present briefly our

*See page 199 for article by Chamberlain and Ryan.

results during the last three years with approximately seventy-five anatomic segmental resections, including bisegmental removals. Several points I believe are of importance; we have not attempted to split the middle lobe segmentally, believing that removal of the middle lobe in toto probably does not sacrifice too much lung tissue even though one segment is involved. We have had occasionally some difficulty in attempting to determine both preoperatively and at operation whether one or several basal segments were involved, and so in general we have performed a combined basal segmental resection. On several occasions we have removed subsegments which seemed to be worth while, and I would like to show two slides to illustrate this. (Slides.)

These lateral films were taken one year apart. In the interim resection on the left side was performed. The original bronchogram showed middle lobe disease and a small amount of disease in the anterior ramus of the anterior segment of the upper lobe. One year later there appears to be some improvement in the middle lobe although there is as much bronchiectasis in the anterior ramus, the small puddle of Lipiodol being in the anterior subsegment. Here is the small lymph node often seen, causing some compression and stenosis, and here the atelectatic portion of the anterior subsegment. The dissection continued with the anterior subsegmental bronchus divided and the lateral subsegmental bronchus left intact.

We have closed the pleural surface because we have had too many complications, we believe, by leaving a raw lung surface. This is performed with little or no actual diminution in breathing capacity, because we do it with the lobe partly expanded, leaving a small rim of pleura which can be brought over.

Here is a patient who has bronchiectasis; this shows the first bronchogram with superior segmental disease, but two areas which are not filled. Further bronchograms were necessary, which showed disease in the posterior upper lobe segment and also in the middle lobe. At operation the distribution was right middle lobe, the superior segment of the right lower, and lateral subsegment of the posterior segment of the right upper. These three areas of disease were removed, resulting in considerably better functional result than if entire lobes were removed.

In conformity with other experience, our complications have been definitely greater, approximately three times greater in the segmental resections than in lobar resections. In particular, empyemas have caused us some difficulty. Whereas in lobectomy there were approximately 3 per cent complications, in the first thirty segmental resections in which a raw lung surface was left we had a 23 per cent rate of empyema, an incidence which we believe to be too high. For that reason we have tended to close practically all raw lung surfaces and in the last 40 odd cases, empyema has dropped to about 5 per cent.

DR. ROBERT R. SHAW, Dallas.—I have enjoyed these two papers very much. Dr. Paulson and I want to report our experience in this field in the last three years. During this period we have done segmental resections for nonmalignant disease in the lung in eighty-four instances and have had a general empyema rate in this whole group of a little more than 16 per cent. This included, however, twenty-two segmental resections done for abscesses and infected cysts. In this group the empyema rate was quite high—34 per cent. If we limit the discussion to patients having bronchiectasis and chronic pneumonitis, there were 60 segmental resections with an empyema rate of approximately 13 per cent. This contrasted with pneumonectomies or lobectomies for the same condition with an empyema rate of 2.7 per cent in 106 instances. There is one other technical aspect of the problem that I would like to emphasize; that is the fate of the remaining segment. The fate of the remaining segment disturbed me considerably early in our experience, especially after the basal segments had been removed from a lower lobe. I noticed on postoperative bronchoscopy that there was kinking of the dorsal bronchus by dislocation of the dorsal segment. Since that time I have routinely left adhesions if they were there, to allow this segment to come down uniformly with the upper lobe in the re-expansion process. If there were no adhesions I made a row of mattress sutures, picking up just the visceral pleural edges so that the dorsal lobe would expand evenly with the upper lobe.

I would like to show one slide to illustrate a problem brought up by segmental resection. In the patient with bilateral bronchiectasis, many more of course are being accepted for surgery now than could have been accepted before segmental resection had become safer. I think the choice of the side to be attacked first sometimes becomes a great problem. We have here an illustration of a woman who has bronchiectasis of the lower segments of the right lower lobe; the lateral film shows that the dorsal segment is normal; there is bronchiectasis complete in the middle lobe, and bronchiectasis in the anterior segment of the upper lobe. To attack the right side first entails a rather difficult dissection and also invites complications that may lead to poor function of the remaining segments in this lung. If a poor function did result on this side it might be impossible to do anything about the lingular segment, which is the only portion involved on the left side. I think it would be better wisdom in a case of this sort to attack the lesser side first, since we would have a functioning right lung—although it is damaged by disease—to give support during the attack on the small lingular segment. I think the left side could be handled easily without too much danger from the operation. Then, with a left lung free of disease, I think the right side could be attacked much more safely. In a later paper you will note that the four deaths occurring in bilateral bronchiectasis occurred after the attack on the second side, and quite often these deaths are due to insufficient pulmonary reserve. If technical difficulties should be encountered on the right side, as sometimes occurs—sometimes an artery is torn so that more tissue has to be removed than was planned—even a pneumonectomy could be performed on the right side and the result would probably be good. If such a thing should occur by attacking the right side first, nothing could ever be done about the disease on the left. It might take a bit of salesmanship to sell the patient the idea of attacking the lesser side first, but I think there are instances in which this would be good judgment.

DR. D. A. MULVIHILL, New York.—I think Dr. Chamberlain has been altogether too modest in glossing over in half a sentence the thirty cases of segmental resection he has done for pulmonary tuberculosis. He has undertaken this study as part of an experimental project at the Triboro Hospital in New York and the U. S. Naval Hospital at St. Albans, N. Y. I have had an opportunity to observe his results to date on this work. He is naturally reluctant to talk about them because he feels, I think, that his indications for segmental resection in tuberculosis are not adequately defined at present and he would like to work them out better. It is true, as he mentioned, that it will take five years to evaluate the final results in these cases. However, thirty cases of segmental resection for pulmonary tuberculosis at this date is no small number. I can say that his results, so far as the early period is concerned, are most promising. He has a low rate of immediate postoperative complications, and so far very few and insignificant late complications in the months that have passed since operation on this group of segmental resections. I think probably he should speak more freely about his experience with this particular group when he closes the discussion.

DR. R. P. GLOVER, Philadelphia.—I should like to re-emphasize one point in the technique of segmental resection as this procedure is applied in tuberculosis. Whereas in nonspecific inflammatory segmental resections we have had excellent success using the technique described by Dr. Clagett, that of leaving the raw surface open and unencumbered by pleura, in tuberculosis the multiple so-called alveolar leaks which frequently occur after segmental resection must be treated with more respect. These leaks ordinarily quickly seal themselves, but in a series of some thirty odd segmental resections in pulmonary tuberculosis we have found that such leaks not infrequently do not become sealed and therefore lead to nonexpansion of the lung. I should like to suggest, therefore, that very meticulous care be taken in tuberculous segmental resections to find and close these leaks, which actually may be tiny bronchiolar openings rather than so-called alveolar tears. These openings must be carefully closed, either by the technique described here or by the

use of a flap of pleura. If this precaution is not taken, proper expansion of the lung will not occur, almost invariably leading to the formation of infected pleural fluid and eventual empyema with bronchopleural fistula. Attention to this detail will give better results in tuberculous cases treated by segmental resection.

DR. J. D. MURPHY, Oteen, N. C.—A few weeks ago at a meeting in North Carolina I was seated next to Dr. Stuart Willis, and the subject of segmental resection in tuberculosis arose. Dr. Willis said it was a vicious procedure and that the results with it were bad. He asked whether we were doing it at Oteen. I was rather noncomittal but when I got home I went through our cases and found that in a series of 110 resections for tuberculosis we had done segmental resection in 9 instances. These varied from a formal dissection of the dorsal segment or an apical segment to simple ligation and excision of the tip of the dorsal segment as described by Dr. Bloch in 1881.

In analyzing the results in these patients, eight had a negative sputum, and reexpansion without complications. One had late progression of the disease into the remaining segment of the upper lobe and required thoracoplasty. All have been done for at least six months and some of them for eighteen months.

DR. FELIX HUGHES, JR., Memphis.—This slide shows the distribution of resections of the lung at Kennedy Hospital since June, 1946. In tuberculosis we have done seventeen segmentals, in bronchiectasis seventeen, including the patients who had both a lobe and a segment removed; for abscess and pneumonitis we have done two segmental resections; for cysts or blebs we have also done two segmentals. In addition, I have listed, under "lesion," patients who have not had a formal segmental resection but have had an excision; there are fifteen of those. There has been no mortality in the group with benign lesions.

In the patients with tuberculosis our results have been more an indication of the stability of the lesion than of the type of resection. Where the patient had a stable lesion, 80 per cent are in good condition at this time, most of them seven to fifteen months after operation. Where the lesion was acute or unstable, only 25 per cent are in excellent condition at this time, and that has been about the same regardless of whether the operation was a lobectomy or a segmental resection. Of the forty-three patients with pulmonary tuberculosis who have had a resection only one patient is in serious condition at this time.

The slides will show the result in two cases. This is a Negro patient with a tuberculous lesion in the right upper lobe, treated by streptomycin. While streptomycin was being given the process spread to two basal segments and the apical segment of the right lower lobe. Lobectomy and segmental resection were performed, with streptomycin protection, and, following this, recovery has been uneventful. Thoracoplasty was done at the end of three weeks, and this slide shows the result after thoracoplasty and resection. It has been over one year since the last operation, and the sputum is still negative.

The next slide shows a patient who had bilateral pulmonary tuberculosis; he had a pneumothorax on the left side and pneumonolysis with complete division of adhesions, but the disease progressed with cavity rupture, a bronchopleural fistula, and total empyema on the left. In addition, therapeutic pneumothorax was instituted on the right, with the diseased area becoming considerably fibrosed during streptomycin therapy. Decortication was done with segmental resection of the posterior apical segment of the left upper lobe, followed, after three weeks, by thoracoplasty. The next slide shows the condition soon after thoracoplasty with operative edema still present. Since that time, this has cleared and the anterior segment is expanded. The sputum is negative and the left lung remains clear. There is a suspicious lesion on the right, but the patient's condition is good.

I agree with Dr. Chamberlain that it is too early to decide the value of segmental resection in tuberculosis, but it is certainly worth considering if we can remove all the diseased tissue and still conserve good lung tissue.

DR. JOHN M. CHAMBERLAIN, New York.—I do not believe that I can overemphasize the vigilance which these patients require postoperatively. When Dr. Samson and Dr. Shaw reported their empyema rate I was rather disturbed and hurriedly went back over my statistics to see if I had been inaccurate in my evaluation. I found that we have recorded one case of empyema in the series, but under a separate heading we have recorded six cases of bronchopleural fistula and under another heading three cases of recurrent or spontaneous pneumothorax. If all are considered to be empyemas, the empyema figure adds up to ten. Yet only one of them developed the clinical picture of an empyema and all of them responded to intercostal decompression (tube) done in the patient's bed without returning to the operating room. I should like to point out that perhaps the great difference in our results is due to postoperative intrapleural active suction. At Bellevue, on Dr. Berry's service, there are eight of us, and when we first began to perform this operation there were two schools of thought as to whether a water seal was sufficient or whether we should use active suction. I believe I can say that most of the men now believe that active intrapleural suction is a better solution.

We have used in one case as high as six intercostal tubes postoperatively (not all at once of course) in instances where localized pockets were found in the postoperative x-ray films. We feel that the dead space is a potential empyema and that its obliteration prevents the development of an empyema. Therefore any intrapleural pockets seen in the early postoperative course of these patients are "tubed" immediately and the tube is left in for twenty-four to forty-eight hours. We wish to point out that if the empyema rate is high it may be because the surgeon failed to insert a tube and re-expand these small pockets which may occur. Perhaps the difference in our results is dependent upon one's definition of what is and is not an empyema.

If I were going to close the surface of one of these segmental resections I cannot imagine a more ingenious way than the one which Dr. Samson has shown us. I have thought for a long time of some way of using a pleural graft, as I saw Dr. Churchill do years ago over the raw area of the mediastinum after a pneumonectomy. Dr. Samson's solution is a good one because as the visceral pleura is approached it should be very easy to skim the visceral pleura off the diseased segment and allow it to fall back over the raw area as he pointed out. However, the collection of air beneath this pleural graft might prevent it from closing the small fistulas. I prefer to rely on suction which removes the air faster than these leaks can deliver it.

I appreciate all that the various discussors have contributed. With reference to tuberculosis I am still not anxious to draw any conclusions. It might interest you to notice the complications we have listed in those 30 cases. Contralateral spreads have occurred in one case; homolateral spreads have occurred in two cases; bronchopleural fistula in three cases; empyema without bronchopleural fistula in none (two have developed since this paper was presented); incomplete or delayed re-expansion in two cases; atelectasis in one case; recurrent spontaneous pneumothorax in none; coronary occlusion in one case; wound infection in no cases.

I do not think anyone can feel that segmental resection in tuberculosis is a matter that can be decided on today, and with Dr. Murphy and the doctor to whom he referred I agree that it is a very hazardous procedure and, first of all, the surgeon should master the technique of segmental resection and then, with the aid of the medical service, consider very cautiously and select very carefully those cases which might be vulnerable to this type of procedure.

DR. FRANCIS M. WOODS, Brookline, Mass.—We agree heartily with what Dr. Chamberlain has said about active suction; we have found that active suction rather than just underwater-seal drainage is definitely more advantageous in getting prompt and complete re-expansion of the lung.

Regarding Dr. Shaw's remarks about the selection of the side for first operation in bilateral bronchiectasis, I would not agree with him because it seems to me that the most important consideration is the postoperative care of the patient and when the side of the

greater bronchiectasis is left in, we often have great difficulty in managing the aspiration and coughing up of secretions. Therefore I would feel at the present time that operating on the side of the greater bronchiectasis is usually more advantageous.

(Slides). This illustrates a complication that Dr. Ramsay did not go into; we have found in two instances, we believe (certainly one was proved at autopsy), that there was considerable engorgement of the superior segment of the lower lobe after the basal segments had been removed. We attribute this, as we attempt to demonstrate in this diagram, to the fact that during exposure of the bronchus to the basal segment of the lower lobe, undue traction or too prolonged traction was placed on the vein to the superior segment. We therefore recommend and practice at the present time, approaching the bronchus from the inferior or anterior side rather than the posterior side, so that undue and prolonged traction will not be placed on that vein.

I would also like to mention that we have become interested in another application of segmental resection. When the chest is opened and the exact diagnosis has not been established, sometimes one finds it difficult to get an adequate biopsy without cutting through lung tissue. In these instances we have done segmental resection to establish the diagnosis. This case turned out to be a carcinoma, and pneumonectomy was completed. The next slide shows a tuberculoma removed by segmental resection. Frozen section established the diagnosis and the chest was closed.

BRONCHOLITHIASIS

HERBERT W. SCHMIDT, M.D.* (BY INVITATION), O. THERON CLAGETT, M.D.,† AND JOHN R. MCDONALD, M.D.‡
ROCHESTER, MINN.

THE subject of broncholithiasis has been of great interest to the medical profession for centuries. It has been said that pulmonary calcifications interested Aristotle (384-322 B.C.).[1] However, a detailed description of the condition did not appear until 1600 A.D. when Schenck a Grafenberg[2] described 17 cases of broncholithiasis reported in the literature. Reports of an additional 6 cases were reviewed by Schenck a Grafenberg, but from the text it is impossible to be certain that the authors were dealing with broncholiths.

Morton,[3] in 1689, outlined therapy for broncholithiasis. He stated:

> First; in the beginning of the disease lubricating medicines should be used copiously; yes, and mild emetics, by which one attempts to promote the expulsion of the calculus or of other bodies that have lodged in the lungs, since long delay makes it more difficult to expel them and the condition of the lungs is thence more gravely injured. Secondly; if, however, severe and spasmodic pains are excited by the laceration of the lungs caused by motion of prickly calculi of this kind, copious hemoptysis is to be expected; and therefore instead of lubricating medicines laudanum should be given freely and frequently, by which the motion of the calculus can be stopped promptly, pain can be relieved and hemoptysis can be checked. . . . Certainly if phthisical patients of this kind take care of their health, as by always breathing pure country air, avoid taking cold, drinking bouts, excessive exercise and whatever else can excite the fixed calculi to new movement, they will be able to live to an advanced old age, performing their usual duties satisfactorily. . . . Only when there is urgent need should a milk diet be ordered in phthisis arising from pulmonary calculi, because such a diet is likely to generate new chalky stones of this kind in the lungs. Whence it happens that the cure of one phthisis is accustomed to lay the foundation for another.

He reported 2 cases of broncholithiasis.

Boerhaave,[4] in 1744, described the case of the botanist, Sebastion Vaillant, who, through many years, coughed up 400 broncholiths and died at 54 years of age of "phthisis calculeuse."

Morgagni[1] stated that "almost 100 authors have described calculi, or tophi, that have been brought up by coughing, or found in the lung." He did not indicate how many of these were bronchial calculi. He again brought up the subject as to whether milk should be used in patients who had broncholithiasis. He agreed with Morton, feeling that it was contraindicated. He quoted other authors, among them an Albertus Fabrius, who contended "very resolutely" that calcareous concretions in the lung might be increased by the use of milk. He cited the case of a patient who craved milk "to such an extent that he drank it even instead of water or beer." It was felt that this program

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"increased the calculus, or at least the pain, and hastened the patient's death." Another patient described by Fabrius was refused milk in the diet and given oil of almonds. A broncholith was coughed up, the pain left, and "perfect health was restored."

In 1810, Bayle[5] described stonelike phthisis as being one of the six types of tuberculosis.

By 1892, microscopic and bacteriologic techniques had been perfected to such an extent that Legry[6] was able to approach the problem from this standpoint. He stated that calcification of tracheobronchial lymph nodes could occur in tuberculous as well as nontuberculous patients. He pointed to the fact that certain workers as early as 1854 had held to this opinion but that their work had not been readily accepted. He stated that tubercle bacilli could be found in only a certain small percentage of the cases in which he studied specimens microscopically. Studies on guinea pigs gave negative results. It was his opinion that this was one way that nature could get rid of a tuberculous lesion, namely, by first having a concretion form and then by having it coughed up. He advised the giving of milk and of calcium phosphate to tuberculous patients since here were substances which would cause a "fossilisation of bacilli and their loss of virulence."

REVIEW OF LITERATURE

Incidence.—The exact incidence of broncholithiasis is difficult to determine because (1) not all of the recognized cases have been reported and (2) not all cases have been recognized. Stivelman[7] stated that the incidence of broncholithiasis in pulmonary tuberculosis is exceptionally low; he saw only 1 instance in more than 5,000 cases of tuberculosis. Pritchard[8] recorded 2 examples as having occurred in 7,000 patients with tuberculosis. Lloyd[9] was able to find reports of only 18 cases of broncholithiasis in the English medical literature from 1900 to 1930. He reported 4 cases of his own. By the end of 1941, VanOrdstrand, Moore, and Harris[10] were able to find reports of 26 cases in the English medical literature. They reported 2 cases at that time. In August, 1944, Tinney and Moersch[11] stated that 30 cases had been reported in the British and American literature. They pointed out that this did not represent the real incidence of broncholithiasis since they were able to collect records of 28 cases from the files of the Mayo Clinic.

Sternberg,[12] in a study of 6,132 post-mortem examinations, observed 32 instances in which there was evidence of perforation into the tracheobronchial tree by an anthracotic lymph node.

By the latter part of 1946, Zahn[13] was able to find reports of 71 cases in the literature. He added another case. His was the only case of broncholithiasis encountered in more than 4,000 cases of tuberculosis during the year 1945 at Fitzsimons General Hospital. Lell,[14] in 1947, reported another 6 cases.

In the previously mentioned reported cases, the diagnosis was made by (1) obtaining a history of having coughed up a broncholith, (2) finding a broncholith at the time of bronchoscopy, (3) surgical exploration, or (4) post-mortem examination.

Etiologic Factors.—Dystrophic calcification was described by Best and Taylor[15] as being "a deposition of calcium salts in dead, dying or chronically inflamed tissues and in areas of fatty or hyaline degeneration." Regions of necrosis, infarcts, scar tissue, caseous tuberculous lesions, hematomas, and degenerated nerve cells may undergo calcification. The factors determining the deposition of calcium salts in devitalized tissue are obscure. It has been suggested that since the carbon dioxide production in such tissue is minimal or entirely absent, the tissue is apt to have a more alkaline reaction than normal which would tend to cause deposition of calcium salts.

Wells[16] pointed to the close analogy between calcification and ossification. He indicated that the processes differ only in their morphologic aspects. The calcium deposits in calculi are first granular but later become homogenized by fusion. He also stated that such deposits usually have no living cells and that no further change occurs unless there is either absorption or addition of more calcium salts. In normal ossification the homogeneous deposits of calcium are closely related to living cells which determine the form of the deposits but which are also able to dissolve calcium salts or cause their deposition as needed.

Chemically, with very few exceptions, the pathologic deposits of calcium have been found to be similar to normal bone, being composed of from 85 to 90 per cent of calcium phosphate and 10 to 15 per cent of calcium carbonate. Wells quoted Poscharissky as having found bone in 60 per cent of the calcified nodules taken from the lungs of 28 persons.

McPhedran[17] stated that osseous calcification may occur in the bronchial cartilages after prolonged suppuration as in bronchiectasis or pulmonary tuberculosis. It may occur after any type of inflammatory process which involves the lung. Thus, calcification of the lungs and the tracheobronchial lymph nodes has been observed following pulmonary infarction and bronchopneumonia and in pulmonary cysts, intrathoracic tumors, actinomycotic lesions, pulmonary abscesses, and the lesions of histoplasmosis. It is curious that broncholithiasis has rarely, if ever, been found in a patient with silicosis.[7] It has been quite generally believed that tuberculosis is the most common cause of calcification of the pulmonary and tracheobronchial lymph nodes. Bech[18] found that in 8 of 26 reported cases of broncholithiasis in the literature, the patient had tuberculosis. For years it has been debated as to what percentage of calcified nodules in the lung and the tracheobronchial lymph nodes harbor viable tubercle bacilli. Feldman and Baggenstoss[19] found 1 positive result in 68 cases in which death was due to other causes than tuberculosis, when they cultured and inoculated guinea pigs with material taken from calcified hilar lymph nodes and Ghon tubercles.

Other workers have stated that in their experience active tuberculosis is not an infrequent finding in the region of lymph drainage between the hilum and the venous angle in the presence of completely calcified or ossified primary tuberculous lesions. From these foci they maintain that reinfection may occur.

It might be well to mention metastatic calcification of the lung. Best and Taylor stated that this is a condition wherein there is a translocation of the calcium of the skeleton to the soft tissues. It occurs when excessive doses of parathyroid extract or irradiated ergosterol have been used. The calcareous deposits may occur in any of the soft tissues. However, the kidneys, arteries, lungs, and the fundus of the stomach are the most common parts that are involved. The kidneys, the stomach, and the lungs are organs that eliminate acid, and theoretically, at least, the cells of these organs might be left more alkaline in reaction. A situation favorable for the deposition of calcium is created. Metastatic calcification may occur in hyperparathyroidism, renal rickets, diseases of bone such as multiple myeloma and osteomalacia, and malignant bone tumors.

It is possible that the phenomenon of metastatic calcification is due to excess calcium in the blood. It has been suggested[15] that, in some instances, such calcification is secondary to tissue injury and therefore is, in reality, a type of dystrophic calcification induced by a toxic agent. It has been shown that parathyroid extract and irradiated ergosterol have a definite toxic reaction other than their effect on calcium metabolism. In some instances, metastatic calcification is associated with hypocalcemia.

Wells[20] said that when metastatic calcification accompanies destructive disease of bone, calcium salts are absorbed in greater amounts than can be held in solution by the arterial blood. Under these circumstances no calcium is found to be deposited on the right side of the heart. After the blood has passed through the lungs and has lost a large part of its carbon dioxide, the calcium salts may be found precipitated in the pulmonary veins and the left side of the heart. The lungs are also a site of calcium deposition under these circumstances. No cellular action has been demonstrated to account for the deposition of these calcium salts. It has been felt that the deposition of calcium salts in tracheobronchial lymph nodes is probably due to a process of filtration alone. We have not observed an instance of broncholithiasis which has occurred secondary to metastatic calcification.

Pathologic Aspects.—Poulalion[21] was one of the first to describe the pathologic features of broncholithiasis. He stated that broncholiths may be cartilaginous, calcareous, or osseous in their make-up and that they may arise anywhere in the pleuropulmonary tissues. Pendergrass and de Lorimier,[22] in attempting to explain the pathogenesis of pulmonary calcification, postulated four possibilities: (1) secondary calcification after degeneration or necrosis due to infection, (2) primary or secondary calcification of aspirated foreign bodies, (3) precipitation of the osseous inorganic elements due to excessive concentrations of them in the blood, and (4) metastasis of bone-forming cells.

It has been said that broncholiths may originate (1) in the lumen of the bronchus, (2) in the bronchial wall, or (3) around the bronchus and subsequently erode through the wall of the bronchus. It has been stated that aspirated foreign bodies and endobronchial secretion may act as nuclei for the deposition of calcium.[11] Inflammatory processes in the tracheobronchial

tree may lead to necrosis of the tracheobronchial cartilages which likewise may act as nuclei in the formation of broncholiths.

Most workers feel that the great majority of broncholiths arise when tracheobronchial lymph nodes become calcified secondary to an antecedent pulmonary infection. These calcified lymph nodes erode the bronchial wall and gain partial or complete entrance into the bronchial lumen. Such a broncholith acts like a foreign body.

Broncholiths may vary in size from granules to the size of 139 Gm.[9] They usually are gray-white, irregular, and hard, but they may look like putty. They usually are rough and irregular. In most instances, when either coughed up or removed bronchoscopically, they are broken so that they may represent merely a fragment of a large stone. They may show black areas of anthracosis. A broncholith which is not coughed up promptly is quite likely to cause collapse of the lung distal to the site of obstruction. Subsequently, pneumonitis, bronchiectasis, lung abscess, or empyema may follow owing to bronchial obstruction and secondary infection.

Fox and Clerf,[23] using iodized oil, were able to demonstrate cylindrical bronchiectatic lesions distal to the site of obstruction in 4 cases after removal of the broncholith.

DATA ON FORTY-ONE MAYO CLINIC CASES

Cases Included.—Included in this series are the cases in which there was a history that one or more broncholiths had been coughed up but in which, at the time of examination, no broncholith could be found. Included also, of course, were the cases in which a broncholith was found either free or fixed in the lumen of the bronchus at the time of bronchoscopy or surgical treatment. At least a part of the broncholith must have eroded into the lumen of the bronchus before the case was eligible for inclusion. The distinction, we feel, is important since calcified lymph nodes that create only extrinsic pressure on a bronchus should not be designated as broncholiths.

Since the publication of the article by Tinney and Moersch in August, 1944, which was based on 28 cases, an additional 13 cases of broncholithiasis have been encountered at the clinic. It is of interest to note that broncholithiasis was recognized in only 28 patients in the thirty-seven years which preceded August, 1944, but that it has been recognized in 13 patients in the succeeding four years. This rapid increase in incidence has occurred by virtue of the fact that an increasing number of indeterminate pulmonary lesions are being explored surgically at an early date to make certain that they are not bronchiogenic carcinomas. In 8 of the cases of broncholithiasis encountered since 1944, the condition was diagnosed at the time of surgical exploration, and in only 3 was the lesion recognized at the time of bronchoscopy. Exploration carried out in 2 additional patients showed pathologic changes secondary to the presence of broncholiths which had been expectorated prior to operation.

This review covers the entire series of 41 cases encountered at the clinic, including the 28 cases reported by Tinney and Moersch. By basing our analysis on the entire series of cases, we believe that the validity of the statistical data obtained is improved.

Twenty-one of the patients were women and 20 were men. The age distribution was as follows: 20 to 29 years, 1 case; 30 to 39 years, 6 cases; 40 to 49 years, 18 cases; 50 to 59 years, 11 cases; 60 to 69 years, 3 cases, and 70 years or more, 2 cases. Thus, 71 per cent of the cases occurred in the age group of 40 to 59 years.

The diagnoses in these cases previous to examination at the clinic were as follows: tuberculosis, 9 cases; carcinoma, 4; bronchial obstruction with stenosis, 4; bronchiectasis, 3; pneumonia, 2; broncholithiasis, 2; lung abscess, 1; and empyema, 1. No previous diagnosis had been made in the remaining 15 cases.

Symptoms.—Broncholiths are foreign bodies in the tracheobronchial tree and thus are usually very irritating. The severity of symptoms depends on the size and location of the broncholith. The symptoms may vary in degree from an unconscious raising of a small stone with a mild cough to the severe stone asthma or bronchial colic with which there may be intense substernal pain accompanied by a sense of suffocation.

The duration of symptoms in this group of patients was of interest. According to the history, the shortest duration was thirty days, and the longest, twenty-two years. The first symptom was cough in 23 cases (56 per cent), hemoptysis in 9 cases (22 per cent), and thoracic pain in 9 cases (22 per cent).

The most common symptom was cough, which occurred in all cases. The cough was productive of only stony material in 1 case. In 25 cases (61 per cent) there was slight to moderate expectoration. Eight patients (20 per cent) gave a history of coughing up as much as 2 ounces (about 60 c.c.) of mucopurulent secretion in twenty-four hours. Seven (17 per cent) raised from 3 ounces (about 90 c.c.) to more than one cupful (about 240 c.c.) of mucopurulent secretion in twenty-four hours.

The next most frequent symptom was hemoptysis. This occurred in 35 cases (85 per cent). Six of these patients had undergone such severe hemorrhage that they had required transfusion.

Fever, usually in association with chills, had occurred in 25 cases (61 per cent). Pleuritic pain occurred in 23 cases (56 per cent), and at times this pain was severe. The pain may occur in the retrosternal region and extend to the thoracic spinal region. A certain number of patients had an asthmatoid wheeze. At times this was severe, resulting in intractable asthma.

Physical Findings.—The physical findings were not particularly diagnostic in the great majority of the patients. The physical findings depended on the size of the broncholith and its location. If a broncholith was located in a tertiary bronchus, physical findings were sometimes absent. If the broncholith was located in a secondary bronchus and caused a high degree of bronchial obstruction, the physical findings were those seen in collapse of a lobe. Thus, there would be dullness to percussion and decreased or absent breath sounds over the region affected. The mediastinum was displaced toward the affected side and the intercostal spaces were decreased in size. Sometimes there was an inspiratory lag on the affected side and elevation of the diaphragm. One of the earliest findings was usually the reduction of the intensity of breath sounds

over the affected part. Sometimes a wheeze was heard over the region; at times this wheeze was heard only at the end of a deep inspiration.

Roentgenologic Findings.—The roentgenologist suspected broncholithiasis in only 3 of the 41 cases. The roentgenologic diagnoses were as follows: obstructing lesion with atelectasis, 6 cases; bronchiectasis, 4; calcified hilar nodes, 7; "rule out bronchiogenic carcinoma," 6; pulmonary abscess, 5; broncholithiasis, 3; tuberculosis, 3; negative for thoracic disease, 2; infiltration of hilus, 1; multiple areas of pulmonary calcification, 3; and right pneumothorax and fibrosis of both upper lobes, 1. In the last-mentioned case, the patient had had a right artificial pneumothorax induced prior to his arrival at the clinic, in an attempt to control pulmonary hemorrhage.

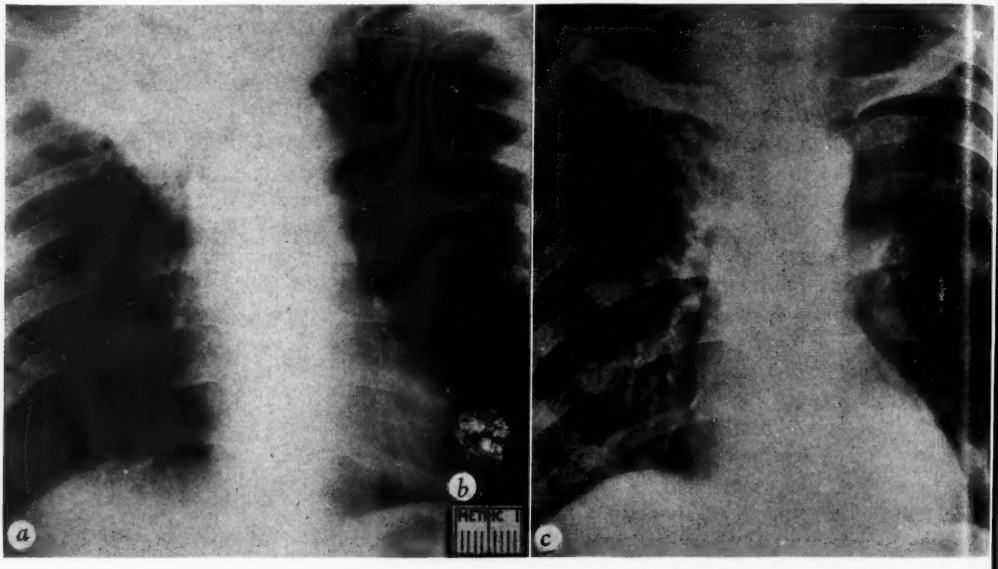


Fig. 1.—*a*, Broncholith occluding right lobe bronchus. The broncholith could be felt with grasping forceps at the time of bronchoscopy but could not be removed. The right upper lobe bronchus was dilated. *b*, The stone that was expectorated immediately after bronchoscopy. *c*, Appearance one month after bronchoscopy. (From Tinney, W. S. and Moersch, H. J.: Broncholithiasis, S. Clin. North America 24: 830-838, 1944.)

In retrospect, probably the most diagnostic roentgenologic finding, when it occurred, was the presence of a dense area of calcium deposition at the apex of a triangular portion of collapsed lung (Fig. 1). The apex of the triangular shadow was located at the mediastinum, and the portion of the lung that was collapsed corresponded to a segment or a lobe. At times this area of calcification was hidden by the heart, and heavy-exposure technique was required to visualize it.

With the collapse of all or a portion of the lung, there may be diminution in the size of the affected pulmonary field. Thus, the diaphragm may be elevated, the mediastinum shifted to the affected side, and the intercostal spaces decreased on this side.

Broncholithiasis may cause pulmonary abscess or empyema, and thus the roentgen findings may be those of these complications. If the broncholith is small, the roentgenologic examination of the thorax may give negative results (Figs. 2 and 3).

Some roentgenograms showed only multiple areas of calcification throughout both lungs. Others revealed the picture of unilateral or bilateral hilar calcification without any evidence of collapsed lung.

Bacteriologic Observations.—The sputum was examined for tubercle bacilli by direct smear in all cases and was found to be free of such microorganisms.

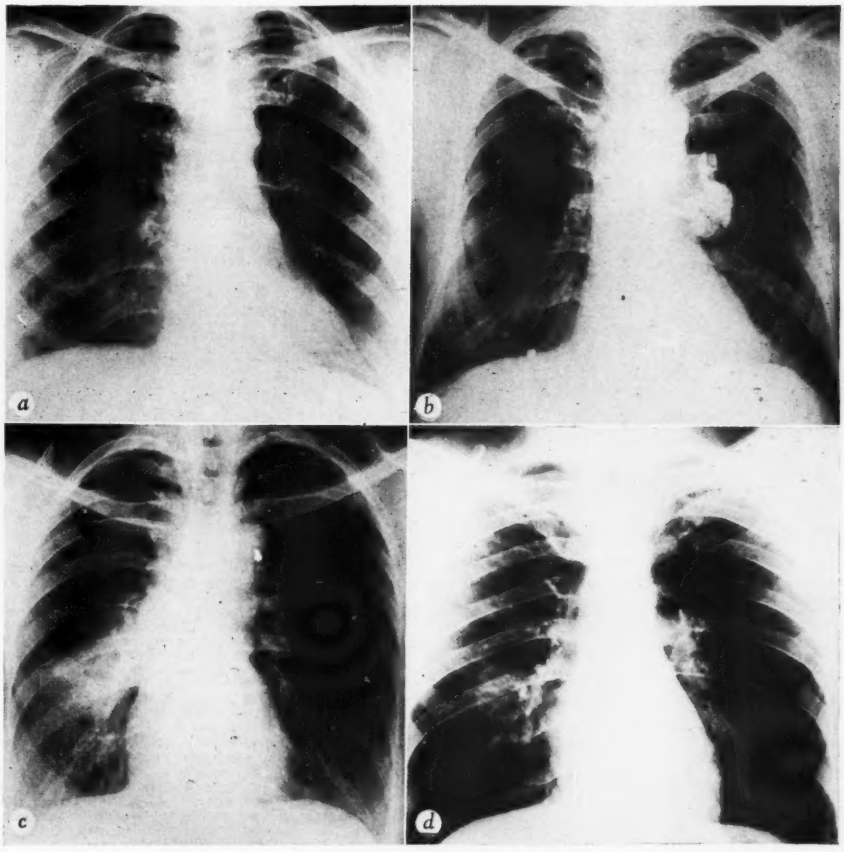
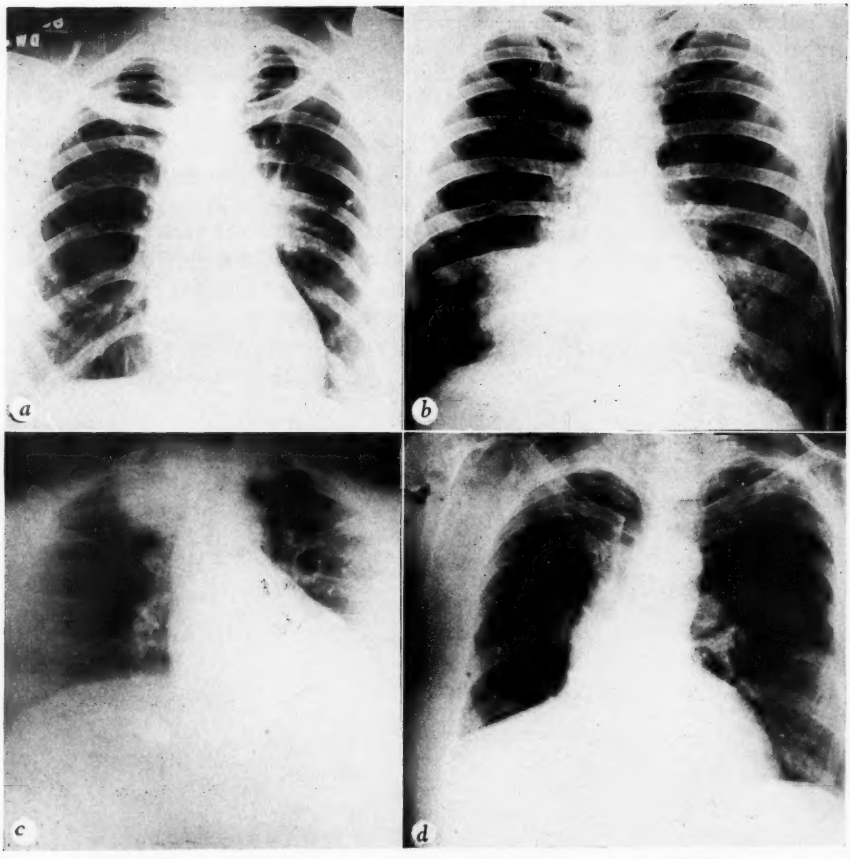


Fig. 2.—Variation in roentgenographic appearance in broncholithiasis. *a*, The appearance is essentially normal yet the bronchoscopist found and removed a broncholith from the left-main bronchus. *b*, Extensive calcification of the tracheobronchial lymph nodes. A broncholith was found and removed from the left lower lobe by the bronchoscopist. *c*, Broncholiths may produce the roentgenographic findings of pulmonary abscess. In this case, a right lower lobectomy was performed. Two broncholiths were found in the depths of the right lower lobe bronchus. Active pulmonary tuberculosis was also found. Guinea pigs inoculated with material taken from the right lower lobe were found to be positive for tuberculosis. *d*, The lesion may resemble those seen in coccidioidomycosis, with a well-circumscribed annular shadow in which there is a central area of rarefaction.



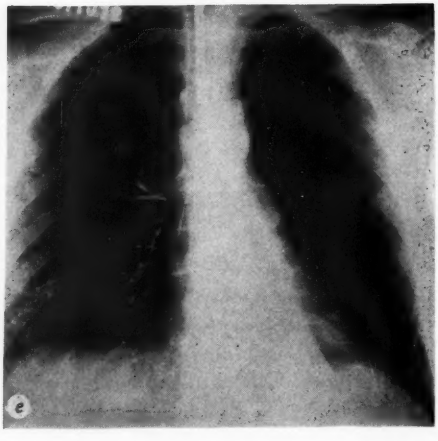


Fig. 3.—The roentgenogram may closely resemble that seen in certain cases of bronchiogenic carcinoma. *a*, An area of infiltration extending from the left hilus into the left upper lobe. Bronchoscopy revealed, in the left upper lobe bronchus, a large broncholith which was removed. *b*, Infiltration into the right middle lobe. Bronchoscopy revealed, in the right middle lobe bronchus, a 0.5 by 0.75 cm. stone which was removed. *c*, Lordotic roentgenogram showing an area of infiltration in the apical segment of the right upper lobe which increased rapidly in size during the month it was followed. A right upper lobectomy was performed. Three broncholiths were found in the excised specimen. *d*, Collapse of the right lower lobe. Bronchoscopic examination revealed, in the right lower lobe, a large broncholith which was removed. *e*, Collapse of the left lower lobe showing as a triangular shadow of increased density behind the heart. Bronchoscopy revealed a mass of tissue in the left lower lobe. The bronchoscopist thought he was dealing with a bronchiogenic carcinoma but the tissue removed showed only inflammatory changes. After bronchoscopy, the patient coughed up a large broncholith. Iodized oil has been placed in the right lung.

In some instances, cultures for tubercle bacilli and fungi were performed with negative results except in 1 instance. In only 1 of the surgical cases was evidence of active tuberculosis found. In this case, guinea pigs inoculated with material from the resected lung were found to be positive for tuberculosis.

Bronchoscopic Data.—After the history, physical examination, and roentgen studies of the thorax and bacteriologic studies of the sputum have been completed, one may or may not think of the possibility of broncholithiasis. Usually, in our series of cases, the clinician considered bronchiogenic carcinoma as being the most likely diagnosis prior to bronchoscopy.

Six of the 41 patients did not undergo bronchoscopy. These were persons who gave a history of having coughed up stones prior to coming to the clinic but who, at the time they were seen, were asymptomatic. Thirty-five patients underwent bronchoscopy one or more times; 32 (91 per cent) of these had positive findings; in 17 instances (48 per cent) the broncholith was either found and removed or coughed up immediately after bronchoscopy. In 1 additional case the broncholith was found but could not be removed. In 10 cases the involved bronchus was found to be strictured or obstructed by granulation tissue.

The bronchoscopic findings often were at first suggestive of a tumor since frequently the broncholith was buried in a fleshy mass of granulation tissue. When this tissue was removed by biopsy forceps, the broncholith was felt or seen. It is extremely important to do a biopsy on this tissue since broncholiths may occur in association with bronchiogenic carcinoma. The surrounding mucous membrane was inflamed, and often a large amount of mucopurulent secretion was dammed up distal to the stone. In 4 cases, bloody secretion was found to be coming from the involved bronchus. In 3 instances bronchoscopy gave negative results.

Once the broncholith is removed, the bronchial mucous membrane usually returns to normal. There is usually no residual bronchial stricture. If bronchiectasis develops distal to the broncholith, the bronchoscopist will see purulent secretion coming from that portion of the bronchial tree distal to the site of the broncholith.

The bronchoscopic examination may not reveal the diagnosis, depending on the location of the stone. If the stone can be seen, it usually can be removed. If it is deeply embedded in an upper lobe bronchus, it may be impossible to grasp it securely enough to remove it. By dilating the bronchus, the broncholith may be coughed up after bronchoscopy.

Two complications which may occur following bronchoscopy should be mentioned. They are (1) hemorrhage and (2) pneumothorax on the involved side.[24] They are not usually serious complications.

Surgical Treatment.—If the broncholith is located in a part of the bronchial tree that cannot be visualized by the bronchoscopist, the problem of a peripherally placed indeterminate bronchial lesion confronts the physician. In our experience it is far safest to explore such a lesion early since a large percentage of these lesions have turned out to be bronchiogenic carcinomas. At the time of exploration the broncholith will be found, and segmental resection, lobectomy, or pneumonectomy may be necessary.

In the future, more and more broncholiths will be found by the thoracic surgeon at the time of thoracotomy. Myers,[25] in reporting 4 cases of broncholithiasis, stated that in 1 of the cases the diagnosis was made when a right lower lobectomy was performed for intractable hemorrhage from this lobe. VanOrdstrand and co-workers,[10] in reporting 2 cases, performed right pneumonectomy for an indeterminate lesion. Before and at the time of the operation, it was believed that a bronchiogenic carcinoma might be present. A calcified nodule was found to be obstructing the bronchus of the right middle lobe by exerting extrinsic pressure. Anderson and MacKay[26] reported a case of an indeterminate lesion involving the right middle lobe. This lesion caused a collapse of the lobe. The bronchoscopist did not make the diagnosis at the time of his examination. A right middle and right lower lobectomy was performed. At this time, 3 calculi were found in the right middle lobe. The calculi had originated in calcified lymph nodes that lay posterior to the bronchus and had eroded through its wall.

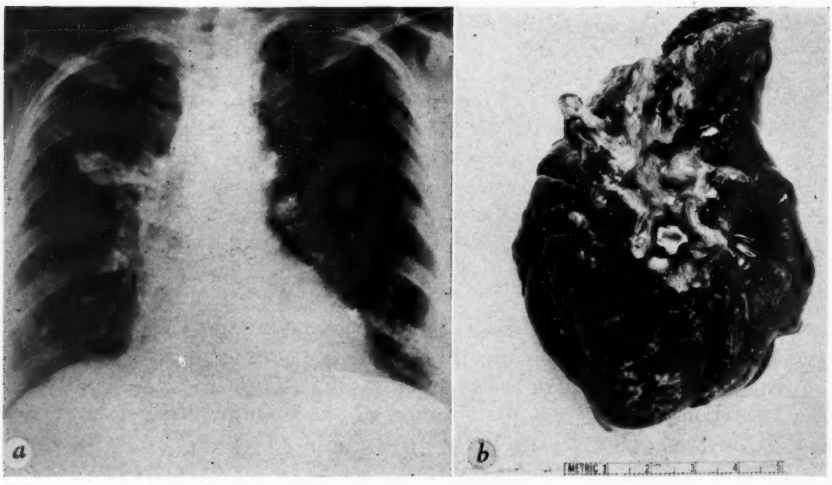


Fig. 4.—*a*, Area of obstructive pneumonitis in the anterior segment of the right upper lobe shown in roentgenographic study. A right upper lobectomy was performed, which revealed an irregular, whitish broncholith in the upper lobe bronchus with marked bronchiectasis distal to the broncholith. *b*, The broncholith.

There were 10 cases in our series in which it was necessary to remove part or all of the lung. Nine of these represented removal of a portion or all of the right lung, and only 1 represented removal of a portion of the left lung. In 2 of these cases the broncholith had been coughed up a short time prior to operation and no broncholith was demonstrated at operation. In these 2 cases, a residual stricture was present which necessitated lobectomy. In both cases, there was marked obstructive pneumonitis in the periphery of the affected lobe. The location of the broncholith in the remaining 8 cases was as follows: 3 were in the upper lobe, 1 was in the middle lobe, 2 were in the main stem bronchus on the right side, and 2 were in the lower lobe bronchus. In 3 instances there was more than 1 broncholith, while in 5 there was only 1 (Fig. 4).

The largest broncholith measured 1.5 cm. in diameter, the smallest 0.3 cm. The broncholiths were more frequently whitish than any other color. A few were yellowish, and others were black, indicating the deposition of carbon pigment within their substance. Broncholiths usually have irregular knobs on them and look much like a staghorn calculus of the kidney in miniature.

There were no surgical deaths in our series. Empyema developed in 1 patient postoperatively, which necessitated performing external drainage. He made an uneventful recovery after this operation.

Nineteen of our patients gave a history of having coughed up multiple broncholiths nonsynchronously. One patient stated that he had coughed up 103 stones. Because of the possibility of multiple sites of origin, we feel that any surgical procedure directed toward the eradication of this type of lesion should be as conservative as possible to preserve the maximal amount of pulmonary function.

Pathologic and Etiologic Features.—Histologic examination of the broncholiths revealed no structure because of cellular death as the result of detachment from their blood supply. The general conclusion reached from the chemical examination of 3 broncholiths in this series is that the chemical constituents are about right for bone (Table I).

TABLE I. COMPOSITION OF BRONCHOLITHS

CASE	WET SAMPLE (MG.)	DRY SAMPLE (MG.)	ASH (MG.)	CALCIUM IN SAMPLE (MG.)	CALCIUM IN ASH (PER CENT)	PHOSPHORUS IN SAMPLE (MG.)	PHOSPHORUS IN ASH (PER CENT)	CALCIUM-PHOSPHORUS RATIO	VOLATILE MATTER (PER CENT)
1	99.1	97.3	83.8	30.5	36.4	14.0	16.7	2.18:1	13.9
2	96.9	93.3	79.6	28.7	36.1	13.3	16.7	2.16:1	14.7
3	106.9	100.6	81.4	28.1	34.5	12.8	15.8	2.2:1	19.1

The causes of calcification in the lung and bronchial lymph nodes are varied. The granulomatous diseases may produce calcification or ossification in the lung and the hilar lymph nodes. The most frequent of these diseases is tuberculosis. Calcification or ossification is common in the Ghon complex, either in the periphery of the lung or the hilar nodes (Fig. 5), but may be seen also in the adult form of tuberculosis. It seems likely to us that the peripheral calcification from any cause is of little importance in the production of broncholiths because it is unrelated to a bronchus which is large enough to permit the nodule to ulcerate into its lumen. On the other hand, it would appear that calcareous or ossified peribronchial lymph nodes, in particular those associated with larger bronchi, are capable of ulcerating into a bronchus and eventually becoming detached. Of the other granulomatous diseases which produce calcification, the most noteworthy is coccidioidomycosis and histoplasmosis.

A second type of calcification (Fig. 6*a*) or ossification (Fig. 6*b*) in the lung is that seen in the elastic cartilage of the bronchi. Cartilage is seen in the wall of bronchi larger than 1 mm. The process of calcification and ossification of the cartilage is unrelated to inflammation but is, rather, a part of the picture of senescence. It is very commonly seen, particularly in older persons. Such calcareous or ossific plaques, when formed in the wall of bronchi, would

Fig. 5.—Calcification of a lymph node subjacent to the main bronchus (hematoxylin and eosin, ×9).

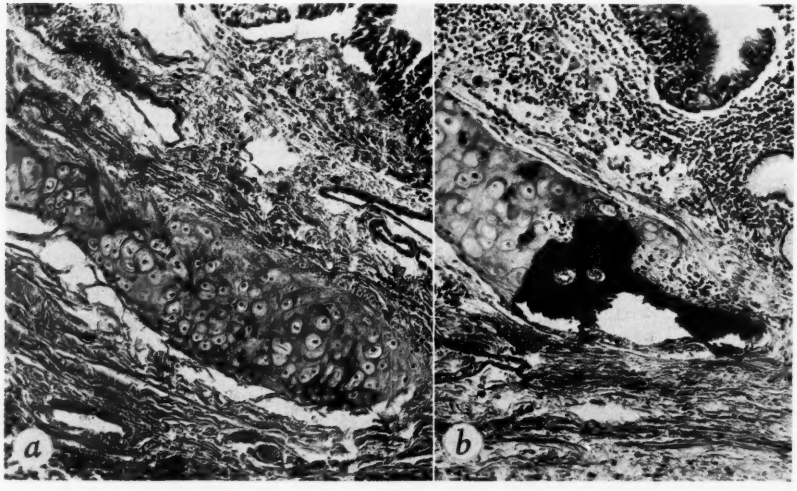


Fig. 6.—*a*, Calcification of bronchial cartilage (hematoxylin and eosin, ×105). *b*, Ossification of bronchial cartilage (hematoxylin and eosin, ×95).

be just as capable of sequestrating into the bronchial lumen as would lymph nodes which had undergone similar changes. As a matter of fact, such plaques in cartilage are originally in the wall of the bronchus while those in lymph nodes are situated outside the bronchial wall. Therefore, those in cartilage would appear to form broncholiths more readily by sequestration because of their proximity to the bronchial lumen than would calcified lymph nodes.

The third type of calcification or ossification is that seen in metastatic calcification. This is the condition in which "calcium salts are deposited in tissues previously normal but with a tendency to alkalinity." Metastatic calcification of the lungs is seen in osseous disease, renal disease, hypervitaminosis D, and hyperparathyroidism. The calcium is deposited usually in the periphery of the lung, in the walls of blood vessels, and in the walls of bronchi. It seems very unlikely that this type of calcification leads to broncholithiasis very often, inasmuch as it is found in the peripheral part of the lung, and such certainly has not been the case in our experience.

Calcification or ossification is also seen in the lung in metastatic sarcoma, particularly osteogenic sarcoma and chondrosarcoma. In addition, hamartomas frequently show calcification or ossification. It is obvious that this bears little relation to the problem of broncholithiasis. Our experience would indicate that the first two processes, namely, calcareous or osseous change in peribronchial lymph nodes subjacent to larger bronchi, as well as calcareous or osseous changes in bronchial cartilages, are the two most important factors in the production of broncholithiasis. Of the 10 cases in which the lung was available for study, ossified or calcareous cartilages were present in 9 cases, ossified or calcareous nodes were present in 5 cases, and in 1 case there was no calcific change in either the bronchial cartilage or the bronchial lymph nodes which were removed with the specimen. It is impossible, of course, in a given case, to be sure which of these two factors played the important role, although in the 5 cases in which calcareous or ossified nodes were present, it seemed to us very likely that the source of the broncholith was a lymph node. In addition, in the 2 cases in which the broncholiths were black, this also would indicate an origin from lymph nodes. The calcified or ossified lymph node or bronchial cartilage appears to act as a foreign body, and it extrudes through the bronchial wall into the lumen. The mucosa of the bronchus at the site of the broncholith is raised into folds and there is subjacent inflammation and granulation tissue (Figs. 7 and 8); as a result, the broncholith is enmeshed so that oftentimes it became invisible on cursory gross examination. It is only after exploring the narrowing of the bronchus that one locates the underlying trouble. As has been seen in 2 cases in which surgical resection was performed even after removal of the broncholith, a permanent stricture may result. The periphery of the lung in all cases showed the condition which has been called "obstructive pneumonitis." In this condition the bronchi are usually dilated, the pulmonary alveoli and bronchi are filled with exudate, and there are varying degrees of fibrosis dependent upon the time during which the obstruction has been operative. This pneumonitis is no different in obstruction caused by broncholithiasis from what it is in any other type of obstruction, and it is for this reason that broncholithiasis simulates carcinoma of the lung.

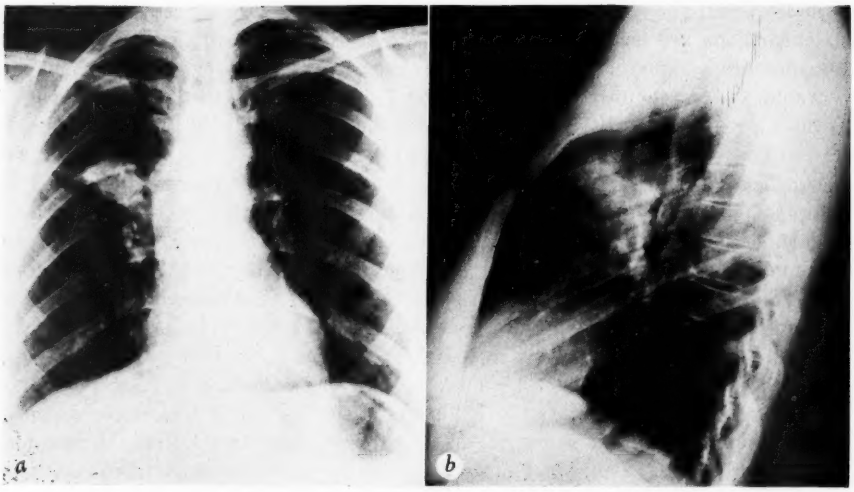


Fig. 7.—Lesions in the anterior segment of the right upper lobe. Bronchoscopy revealed a stenosed bronchus of the right upper lobe. A right upper lobectomy was done. There were multiple broncholiths in the right upper lobe bronchus. *a*, Anteroposterior view, *b*, lateral view.

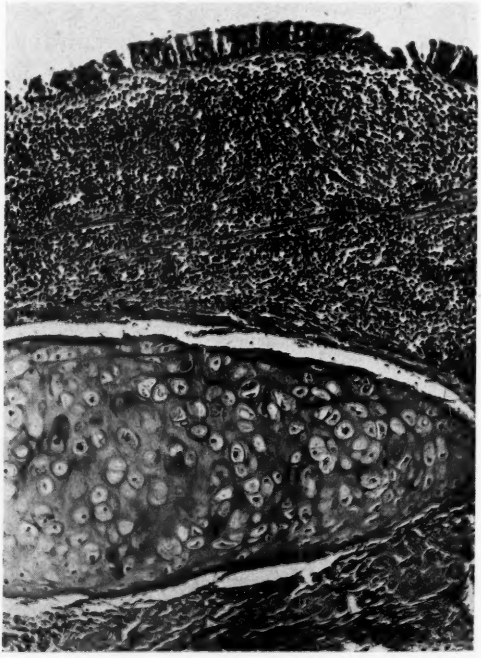


Fig. 8 (Same case in Fig. 7).—Bronchial wall subjacent to a broncholith. Note the inflammatory reaction in the mucosa of the bronchus (hematoxylin and eosin, ×95).

Active tuberculosis was not a feature in the pulmonary tissue under consideration. In only 1 case was active tuberculosis found in the lobe. In this patient the results of guinea pig inoculation were positive. In 1 other case, there were giant cells and a granulomatous reaction around the ossified peribronchial lymph nodes suggestive of activity.

The location of the broncholith could be identified accurately from the records in 28 cases. In 3, the right main bronchus was involved; in 2, the right-stem bronchus contained the broncholith. The location of the rest of the stones was as follows: right upper lobe, 6 cases; right middle lobe, 3; right lower lobe, 6; left main bronchus, 1; left upper lobe, 2; and left lower lobe, 5. The right lung was involved twenty times and the left lung eight times.

If most broncholiths originate outside the bronchial tree in calcified lymph nodes, as we believe they do, one could partly explain the location of the stones by the fact that there are a greater number of lymph nodes associated with the right lung than with the left. There are a greater number of lymph nodes which lie in association with the right upper lobe bronchus than with those of the right middle or lower lobe. Miller[27] stated that lymphoid tissue in the lung increases from childhood to old age. He said that this increase is due largely to the irritation that arises from the constant inhalation of irritating substances.

Follow-up.—In the group of 13 cases in which either bronchoscopy was performed and no evidence of broncholithiasis was seen or in which bronchoscopy was not done because it was not thought to be indicated, 1 patient was in excellent health on follow-up study. One had had no further trouble so far as the lungs were concerned, but died of coronary occlusion at the age of 63 years, twelve years after his visit to the clinic. Four patients raised additional broncholiths after leaving; 1 stated that he had raised 40 stones after his visit, but other than this he felt that he was in good general health. Two patients continued to raise bloody sputum. In 5 instances accurate follow-up information could not be obtained.

The 18 patients who were found to have broncholithiasis at the time of bronchoscopy had the following results: Eleven became asymptomatic after the removal of the broncholith. One patient regarded herself as being in excellent health "except I still spit up" thirteen years after removal of the broncholith. One patient, 64 years of age, stated that he was in excellent health except for "some shortness of breath on exertion" seven and one-half years after removal of a broncholith. One woman, aged 70 years, who had, firmly embedded in the right middle lobe bronchus, a broncholith which could not be removed, stated that she was asymptomatic on our survey. Three patients in this group had poor results. One man had a good immediate result after removal of the broncholith, but his widow recently wrote that "his lung condition gradually grew worse ending in a heart attack and a hemorrhage." Death occurred in this case ten years after the broncholith had been removed. One patient had a contracted left lower lobe which did not expand after a broncholith was coughed up after bronchoscopy. A left lower lobectomy was

advised but the patient refused to have the operation. A lung abscess developed in 1 patient, with a metastatic abscess of the brain following bronchoscopy; he subsequently died.

Eight of the 10 patients who underwent surgical resection for broncholithiasis had excellent results. They have had no further symptoms referable to their lungs. In 1 patient, there developed an empyema which necessitated external drainage. One patient stated that she had "cardiac asthma" and "fluid" on the remaining lung three years after a right pneumonectomy. It was felt that these symptoms were due to inadequate pulmonary function.

SUMMARY

Some of the historical features of broncholithiasis are recorded. Data from the standpoint of its incidence, etiology, and clinical findings are given.

A review of data on 41 patients who were seen at the Mayo Clinic because of this condition is presented. Twenty-one of the patients were women and 20 were men. Seventy-one per cent of the cases occurred in the age group of 40 to 59 years. In 15 cases the diagnosis was made on the basis of a history that the patient had coughed up a broncholith. In 18 cases the diagnosis was made at the time of bronchoscopy, and in 8 it was made at the time of operation. The last-mentioned group of patients underwent exploration because bronchiogenic carcinoma could not be ruled out prior to operation.

Because of the possibility of multiple sites of origin, it is felt that the surgical procedure directed toward the eradication of this type of lesion should be as conservative as possible in order to preserve the maximal amount of pulmonary function.

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DISCUSSION

DR. JEROME HEAD, Chicago.—I have been very interested in this subject for a number of years. Dr. Mohan and I presented a paper on the subject at the Trudeau Society meeting in New York last June, which will be published in the *American Review of Tuberculosis* in the next month or so. I think the condition is extremely common and that one will find it in proportion to the diligence with which he looks for it, as was stated in the paper. These patients frequently do say that they have coughed up stones; they think that is more or less a usual thing with a cough and they do not say anything about it. Unless one asks the patient specifically whether or not he has coughed up stones he often fails to reveal the fact. We had one patient who had had an atelectasis of a lobe; any number of doctors had examined him and questioned him, and when I made rounds I asked, "Have you ever coughed up anything peculiar?" He pulled out his pocketbook and took out an envelope with about 20 stones, which he had not shown to any other doctor—he had to be asked.

This patient had stones and a cancer; the cancer had eaten into a calcified gland and freed calcium. Most cases, of course, are caused by tuberculous calcified glands rupturing into the bronchus. We had one case recently with a typical clinical and x-ray picture of histioplasmosis in which stones were coughed up from a calcified gland in the hilus.

(Slide.) Dr. Mohan became interested in the anatomy of the glands around the roof of the lung and worked out this diagram; this has been done before and his work merely verified what has been done previously. The most important gland, I believe, is No. 4 on the right, which is the bifurcation gland; it runs down along the right-main bronchus. That is absolutely constant. No. 2, No. 1 below the upper lobe, and No. 3 are very important ones just below the middle lobe. The glands on the left side are less constant and less regular and it was pointed out that rupture into the bronchus is rather uncommon on the left side.

This is a patient in whom the gland at the bifurcation ruptured into the right main bronchus and into the esophagus. The patient had empyema and lung abscess which had broken into the pleural cavity. This was drained; Dr. Holinger used bronchoscopy and esophagoscopy, and cauterized the two openings; they healed and the patient recovered without anything but drainage of an empyema. That was many years ago and I think today the appearance of this complication would justify operation.

To show how common it is, in the last six months we have seen 6 cases, and we have under observation 3 patients at the present time in whom we feel sure that a calcified gland is causing recurrent atelectasis, and we anticipate that they will cough up calcium in the

near future. There is no set treatment for the condition. Most patients cough up a stone or two, or three or four. They have an occasional spell of fever and atelectasis, the condition will pass away, and they have no more trouble. Operation should be done only in those cases in which there is continuing and recurring trouble. A period of rest in bed and one month or so of intensive penicillin treatment will often permit the infection in the gland to clear up and the gland will heal over. Removal of the stones at bronchoscopy is relatively dangerous because of the marked tendency to hemorrhage.

DR. EDGAR W. DAVIS, Washington, D. C.—I have enjoyed immensely Dr. McDonald's presentation on broncholithiasis.

In this connection, I wish to report the case of a 50-year-old white man who had been wheezing for about two months prior to the time I saw him. A bronchoscopic examination was performed at which time some granulation tissue was found in the left-stem bronchus. A biopsy of the tissue revealed only inflammatory reaction. The following day a large stone and two small ones were coughed up. The wheezing disappeared, only to recur three months later. Another bronchoscopy was performed, revealing the same findings as on the previous examination. The symptoms of asthma disappeared and reappeared on two other occasions during the following ten months. The fourth bronchoscopic examination revealed a carcinoma in the right lower lobe bronchus, for which surgery was done. It has now been about five months since surgery and there have been no symptoms of asthma.

There is a possibility that some of these other patients with broncholithiasis may develop carcinoma.

DR. E. E. AVERY, Chicago.—I would like to show one slide of a case from Hines Hospital which demonstrates the most conservative surgical management. This 32-year-old patient had a history of recurrent pneumonitis over a period of several years. He had to stop work every two months or so and take penicillin until relieved. The bronchogram illustrates the area of stricture between the right upper and middle lobes which was seen at bronchoscopy; it was a fishmouth type with a very narrow flat orifice which could not be dilated at this time. At surgery, which was performed by Dr. Langston, two possibilities appeared; one was to resect the middle and lower lobes, but it was felt that this would remove a large amount of good functioning lung tissue. Therefore, we decided to excise the stone with its surrounding fibrous tissue and attempt to dilate the bronchus postoperatively. Bronchoscopic dilatation was then accomplished with a very good result.

This case is presented to illustrate the transthoracic excision of a broncholith with postoperative dilatation of the bronchus in an appropriate case.

DR. A. J. GRACE, London, Ont.—I should like to place one case on record. This white woman, 36 years of age, came to me in 1946, with a history of a productive winter cough for twenty years, starting when she was 16. The cough had become persistent all the year round three years before I saw her, and at that time it was more productive of mucopurulent expectoration. Soon after that she began to bring up blood and had constant and repeated hemoptyses for the three-year period. The amount of blood varied from streaking up to what she described as over a cupful of bright red blood, which she felt came from the left side, and she had the feeling that if she exerted pressure on the left side it seemed to help control the bleeding. She also had for one year a history of very severe backache on the left side for which she had consulted an orthopedic surgeon and was given advice and treatment. She felt that the only factors which caused bleeding or aggravated it, were exertion of any kind, jolting or jarring during a rough ride, or anything of that type, and for some reason bleeding was more marked at the time of her menstrual periods. That fact had led a doctor elsewhere to give deep x-ray therapy to the ovaries—a very startling fact when we think of it in terms of the actual pathologic process present.

(Slides.) Investigation along all the usual lines did not give a definite diagnosis, but as the one slide of this chest will show, in the region of the left hilum there is an area of increased density and some calcification. Thoracotomy revealed an area of induration in the apical segment of the left lower lobe, with massive adhesions, indicating an old pleuritic process all the way round, particularly posteriorly, no doubt explaining her back pain. Because of the fact that the bronchogram had suggested some degree of cylindrical bronchiectasis in the basal segment of the lobe, I did a left lower lobectomy. I am glad to say that she made an excellent recovery.

The pathologic report on the specimen described dilatation of the bronchus to the apical segment of the left lower lobe, to form an irregular cavity, some 2½ by 1½ cm., with a rough lining of granulation tissue and complete destruction of the normal epithelium. cm." The summary was, "Broncholith, chronic bronchiectasis."

This cavity contained "a hard calcified dark, calculus-like mass measuring 1 by 0.6 by 0.3

In the last two years she has had no further symptoms and no trouble. I feel that this is perhaps a slightly unusual case; I think, if I interpreted Dr. Schmidt's remarks correctly, he did not have any in the apical segment of the left lower lobe, and the left side is apparently less common. It may be noted that this patient had two calcified glands which were demonstrated at operation, one of which can be seen in the left hilar region. That may well give a clue to the comments Dr. Head has made regarding etiology.

DR. ANDRE COURNAND, New York.—I think that the role of tuberculosis in broncholithiasis should not be minimized. In 1933 with Dr. Oswald Jones we published a paper on "The Shrunken Pulmonary Lobe With Chronic Bronchiectasis" in which it was demonstrated that the retraction of a middle lobe was probably related to the development of a primary tuberculous complex in the following manner: (1) primary parenchymatous lesion; (2) caseous lymph node located around the middle lobe bronchus with invasion of the latter and secondary contraction of the middle lobe. The only clinical evidence of such a process is the presence of calcifications at the hilum, which may eventually be seen to protrude into the lumen of the bronchus. In such a case, which presented a syndrome of a shrunken middle lobe and bronchiectasis without suppuration, the patient coughed up shortly after bronchoscopy two small pieces of calcium, which were no longer visible on the films. Sixteen years later this man 61 years of age was doing very well and, on the basis of that experience, I wonder whether in similar cases the lobe should always be removed.

DR. SCHMIDT.—I wish to thank the discussers. I did not think it would be necessary to try to convince anyone in this group that he should explore peripherally placed indeterminate pulmonary lesions which present the findings of obstructive pneumonitis. I gathered from Dr. Head's discussion that he was somewhat reluctant about exploring this type of lesion. It has been our experience that a fairly large percentage of these lesions have proved to be due to bronchiogenic carcinoma and consequently we have found it far safer to perform exploratory thoracotomy.

In answer to Dr. Cournand's question, it is possible for caseous tuberculous lymph nodes, as well as anthracotic lymph nodes, to erode into the tracheobronchial tree. In a fairly large percentage of cases in which a broncholith has obstructed a bronchus for a long period of time, one finds that bronchiectasis has developed in that portion of the bronchial tree distal to the site of obstruction.

THE RESULTS OF SURGERY IN BRONCHIECTASIS

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DURING the years 1939 to 1945 inclusive, 103 operations for bronchiectasis were performed on 103 patients on the Chest Division of Bellevue Hospital. These cases have been analyzed and grouped according to the extent of the disease process and the type of associated pathology to show the significance of these factors in the surgical treatment of this disease.

Cases in which tuberculosis was demonstrated in the specimen removed have been omitted from this series. Similarly, cases in which the primary disease process was a lung abscess have not been included. Acute suppuration associated with bronchiectasis includes those cases which on microscopic examination revealed active pneumonia and acute inflammation and are differentiated from the group in which chronic inflammatory changes, interstitial fibrosis, plasma cell and lymphocytic infiltration with pneumonitis were present, without evidence of acute inflammation. Cases without acute suppuration are further divided into those with minimal parenchymal changes and those showing marked fibrosis and chronic inflammatory reaction associated with the bronchiectasis. Such pathologic classification has been made to show a possible correlation with clinical bronchiectasis. Clinically it may be impossible to differentiate bronchiectasis with acute suppuration from bronchiectasis with marked chronic inflammatory changes. Our study demonstrates the importance of recognizing the difference in the results of surgery between the two groups, as well as the bearing that the extent of the disease has on the prognosis.

Fifty-five lobectomies were performed on 54 patients for bronchiectasis uncomplicated by acute suppuration, and 30 lobectomies on 28 patients were for bronchiectasis associated with acute suppurative disease. Of the 55 patients without acute suppuration 17 had minimal parenchymal changes in the lung, and 38 had varying degrees of marked fibrosis, chronic inflammation, and interstitial pneumonitis. The remaining 21 patients had bronchiectasis involving the entire lung on one side for which pneumonectomy was performed. Of these, 8 had pathologic evidence of acute suppuration with pneumonia and abscess formation, and 13 had no acute suppurative disease but included cases varying from marked chronic inflammation and fibrosis to minimal parenchymal disease.

One patient with bronchiectasis underwent two lobectomies including the lower lobe on the left side and the lower and middle lobes on the right side. Two patients in the group in which associated acute suppurative disease was present had two lobectomies. In the first patient the upper lobe on the left side was removed followed by the lower on the same side; in the other the right lower lobe was removed in two operations.

Of the resections for uncomplicated bronchiectasis without acute suppuration 5 were for disease in the upper lobes, 31 for disease in the lower lobes, and 3 for disease in the middle lobe. In 15 patients lower and middle lobectomy

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was performed on the right, or lower lobectomy and lingulectomy on the left, and in one patient the right upper and middle lobes were removed at one operation.

For bronchiectasis associated with suppurative pneumonitis lobectomy was performed for disease in the upper lobes in 8 cases, lower lobes in 13 cases, middle lobe in 2 cases. Right upper and middle lobes were removed in 3 instances, right lower and middle in 2 instances, and right upper and part of right lower in 2 instances.

Table I represents an analysis of the mortality in the four groups in which lobectomy was performed in the first two, and pneumonectomy in the third and fourth groups.

The average duration of symptoms in the first group, that is, bronchiectasis uncomplicated by acute suppurative disease, was 9.5 years, indicating that the disease may frequently be of long standing without manifesting symptoms sufficient to bring the patient to surgical intervention. In contrast, in the second group in which bronchiectasis was accompanied by suppurative disease, the symptoms were present for only 2.7 years. In the third and fourth groups in which the disease had progressed to involve the entire lung the symptoms were also present for a longer period in the cases in which no acute suppuration was present than in the group in which the process was more acute.

The age incidence in uncomplicated bronchiectasis was appreciably lower than in bronchiectasis with associated acute suppurative disease, regardless of whether the disease was limited to one lobe or one lung. There was a higher incidence of patients under 30 years of age in cases without acute suppurative disease and a lower incidence above 40 years of age, which included bronchiectasis in one or two lobes or involving the entire lung (Table I).

TABLE I. MORTALITY IN SURGERY FOR BRONCHIECTASIS

PATHOLOGY	NUMBER OF OPERATIONS	AGE GROUPS		AVERAGE DURATION OF SYMPTOMS (YR.)	OPERATION	MORTALITY	
		UNDER 30 YR.	OVER 40 YR.			OPERATIVE* (PER CENT)	HOSPITAL† (PER CENT)
Bronchiectasis in one or two lobes, without acute suppurative disease	55	32	7	9.5	Lobectomy	3.6	5.5
Bronchiectasis in one or two lobes, with acute suppurative disease	30	6	9	2.7	Lobectomy	6.7	10.0
Bronchiectasis without acute suppurative disease, involving an entire lung	13	7	3	7.1	Pneumonectomy	23.1	23.1
Bronchiectasis with acute suppurative disease, involving an entire lung	8	0	5	3.8	Pneumonectomy	37.5	37.5

*Died during first postoperative month.
†Died during prolonged hospital stay.

This table shows that younger individuals are susceptible to infection in the origin of their bronchiectasis. They are, however, less susceptible to acute pulmonary suppuration once the bronchiectasis has developed, and the older the patient the more likely is bronchiectasis to be associated with suppurative disease.

In stating mortality percentages, operative mortality refers to deaths occurring within the first month following operation, and the term hospital mortality refers to all cases in which death occurred during the prolonged hospital stay following operation. This distinction has been made to differentiate the mortality directly referable to operation from that which may have occurred incidental to the disease, such as brain abscess, and that may develop months after operation while the patient is still in the hospital, because, as frequently occurs in city hospitals, he has no other place to go.

It will be noted from Table I that following lobectomy the mortality is almost twice as great in the group in which bronchiectasis is complicated by acute suppuration than in the similar group without suppurative disease. The mortality in these two groups corresponds directly with the incidence of postoperative fistula, pleural infection, and wound infection—in short, with the development of complications. It is important, therefore, in this respect to bear in mind the pathology as directly related to the incidence of complications and hence indirectly to the mortality of resection as an operation for this disease.

Table II shows the causes of death in the four groups in relation to the pathology present and the type of surgery performed. In the first and second groups 33 per cent of deaths were considered due to infections induced by secretions. In the group treated by pneumonectomy half of the deaths were referable to secretions. It is interesting to note that in the first two groups

TABLE II. CAUSES OF DEATH

PATHOLOGY	OPERATION	CAUSE OF DEATH	NUMBER OF CASES
Bronchiectasis in one or two lobes, without acute suppurative disease	Lobectomy	Hemorrhage	1
		Bronchopneumonia	1
		Brain abscess two months postoperatively	1*
Bronchiectasis in one or two lobes, with acute suppurative disease	Lobectomy	Hemorrhage one hour postoperatively	1
		Pneumonia in opposite lung	1
		Brain abscess three months postoperatively	1*
Bronchiectasis involving an entire lung, without acute suppurative disease	Pneumonectomy	Hemorrhage on the table	1
		Anoxia on the table from profuse secretions	1
		Empyema with bronchopleural fistula	1
Bronchiectasis involving an entire lung, associated with abscess, acute suppurative disease, pneumonia	Pneumonectomy	Hemorrhage on the table	1
		Bronchopneumonia in opposite lung	1
		Urinary shut down after transfusion	1

*Hospital mortality.

there was a similarity in the causes of death, namely, hemorrhage, immediate postoperative infection, and late postoperative manifestation of infection (brain abscess) (Table II).

Table III presents an analysis of mortality from postoperative complications in relation to the operative technique. It will be seen that the incidence of bronchopleural fistula following lobectomy is lower in cases in which individual ligation was employed than in those handled by clamp or tourniquet technique. This was true in both bronchiectasis and bronchiectasis associated with suppurative disease. Of all lobectomies performed in this series the incidence of fistula was higher (63.6 per cent) in upper lobectomies than in lower lobectomies (37.8 per cent). In cases in which two lobes or one lobe and a segment of another (lingula) lobe were removed the incidence of fistula was 71.4 per cent.

The higher percentage of fistula in this series has subsequently been lowered because of greater care in handling the stump, more frequent use of individual ligation of the stump with pleuralizing of the bronchial stump and the reduction of the acute suppurative element in the lung that is to be removed before bringing the patient to operation.

TABLE III. MORTALITY FROM POSTOPERATIVE COMPLICATIONS IN RELATION TO TECHNIQUE AND TO EXISTING PATHOLOGY

PATHOLOGY	NUMBER OF CASES	OPERATION	TECHNIQUE		COMPLICATIONS: FISTULA (PER CENT)	COMPLICATIONS: PLEURAL INFECTION (PER CENT)	COMPLICATIONS: WOUND INFECTION (PER CENT)	MORTALITY (PER CENT)
Bronchiectasis in one or two lobes, without acute suppurative disease; with minimal parenchymal changes.	17	Lobectomy	Individual ligation	4	25.0	25.0	0	0
			Clamp	1	100.0	100.0	0	0
			Tourniquet	12	45.5	45.5	18.2	16.7
Bronchiectasis in one or two lobes, with fibrosis of parenchyma and extensive chronic inflammatory changes.	38	Lobectomy	Individual ligation	10	20.0	40.0	20.0	0
			Clamp	7	14.3	28.6	0	0
			Tourniquet	21	57.1	27.1	14.3	4.8
Bronchiectasis in one or two lobes, with acute suppurative disease.	30	Lobectomy	Individual ligation	4	25.0	50.0	25.0	0
			Clamp	7	50.0	100.0	0	14.3
			Tourniquet	19	82.4	94.1	58.8	15.8
Bronchiectasis involving an entire lung, with acute suppuration; abscess; pneumonia.	8	Pneumonectomy	Individual ligation	4	33.3	66.6	0	25.0
			Clamp	2	100.0	100.0	0	50.0
			Tourniquet	2	0	100.0	0	50.0
Bronchiectasis involving an entire lung with chronic infection and marked or minimal parenchymal fibrosis.	13	Pneumonectomy	Individual ligation	6	40.0	60.0	20.0	33.3
			Clamp	0	—	—	—	—
			Tourniquet	7	33.3	33.3	20.0	14.3

Pleural infection following lobectomy in the uncomplicated bronchiectasis group was highest when treated by tourniquet and in bronchiectasis associated with suppurative disease was lowest when treated by individual ligation. Wound infection was highest in all patients with suppuration operated on by both lobectomy and pneumonectomy, in which the tourniquet technique was used. In the pneumonectomy group the incidence of fistula and pleural infection ran parallel, as might be expected. This group represents extremely serious cases in which the amount of sputum at operation averaged about 100 c.c., a reduction from an average of 215 c.c. on admission. Some of these patients were so desperately ill that surgery was used as a last resort in an attempt to interrupt a cycle in which the patient's condition not only became progressively worse over a period averaging six years, but he came to surgery in a condition of malnutrition, wasting, and chronic sepsis from which he could not recover without surgical intervention. These patients, therefore, constitute largely a salvage group and differ from those cases in which the disease had not reached the extent of that in the pneumonectomy group (Table III).

These three groups were also studied in relation to the incidence of sputum as presented in Table IV. It will be noted that in the bronchiectasis group with acute suppuration treated by lobectomy and in the bronchiectasis group without acute suppuration treated by lobectomy, the amount of sputum on admission was approximately the same, but in the second group it was possible before operation to reduce the amount of sputum to almost one-half of that in the first group. Also, the amount of sputum in the cases in which bronchiectasis involved an entire lung was more on admission and at operation than in those in which similar disease was localized to part of the lung. Average sputum on admission of patients who died following pneumonectomy was 400 c.c. daily, which contrasted markedly with 130 c.c. daily admission sputum in the group that survived. In these cases sputum was reduced to 130 c.c. and 85 c.c., respectively, at the time of operation. The absence of acute suppuration, therefore, may best be determined by the relative sputum reduction following preoperative treatment rather than the amount that is present on admission (Table IV).

The practical application of this observation includes delaying resection until the sputum is brought to the minimum in order to reduce the acute suppurative element as much as possible. Preoperative measures included postural drainage with bronchoscopic aspiration in resistant cases. Chemotherapy was employed to reduce secretions and was determined by the type of bacterial flora present in cultures. Sulfadiazine was used at first; later it was replaced by penicillin, as being more specific for more organisms and less toxic for the patient. Recently streptomycin has been added to the specific drugs that may be employed. It is reserved for patients having predominantly gram-negative organisms that are streptomycin sensitive. Patients in whom the organisms present are penicillin resistant are now treated with sulfadiazine. Patients who become allergic to sulfadiazine may be treated with sulfathiazole to avoid the specific sensitivity. Cases of overwhelming anaerobic infection frequently will

TABLE IV. INCIDENCE OF SPUTUM IN RELATION TO MORTALITY

PATHOLOGY	NUMBER OF OPERATIONS	OPERATION	AVERAGE AMOUNT OF SPUTUM ON ADMISSION (C.C.)	TYPE OF SPUTUM BEFORE OPERATION				AVERAGE AMOUNT OF SPUTUM AT OPERATION (C.C.)	HOSPITAL MORTALITY			
				FOUL		NONFOUL			FOUL		NONFOUL	
				NUMBER	PER CENT	NUMBER	PER CENT		NUMBER	PER CENT	NUMBER	PER CENT
Bronchiectasis without acute suppurative disease in one or two lobes	55	Lobectomy	120	20	36.4	35	63.6	40	1	5.0	2	5.7
Bronchiectasis with acute suppurative disease in one or two lobes	30	Lobectomy	125	16	59.3	11	40.7	70	2	12.5	1	9.1
Bronchiectasis without acute suppurative disease involving an entire lung	13	Pneumonectomy	210	7	53.8	6	46.2	80	3	42.9	0	0
Bronchiectasis with acute suppurative disease, involving an entire lung	8	Pneumonectomy	220	7	87.5	1	12.5	135	3	42.9	0	0

respond to oxygen therapy administered with high concentration mask so as to reduce the sputum and convert it from foul to nonfoul variety, that is, from aerobic to anaerobic flora.

Aerosol administration of penicillin or streptomycin has been found of value in reducing secretions when intramuscular injection did not bring about a high enough concentration to affect the organisms. This may be used in conjunction with intramuscular therapy.

The incidence of foul or occasionally foul sputum was found to be lowest in the uncomplicated bronchiectasis group, becoming progressively greater depending upon the presence of suppuration and the degree of lung involvement. In analyzing the mortality in these cases it was found that there was almost no difference between the percentage of deaths in cases with foul sputum and those with nonfoul sputum in simple uncomplicated bronchiectasis. With the presence of suppuration, however, the mortality became appreciably greater in cases with foul sputum, so that when the entire lung was involved the mortality was limited to those patients having foul sputum.

In a total of 106 operations on 103 patients for bronchiectasis there were 12 deaths from various causes as explained in Tables I through IV. Seventy-eight of the 91 survivals were observed over an average follow-up period of four years, and grouped according to original pathology.

Of the 78 cases followed, 65 were lobectomies and 13 were pneumonectomies. Of the 65 lobectomies, 16 patients had other lobe involvement with bronchiectasis. Twelve of these were not resected at a later date, and 2 patients died; 4 had further surgery for other lobe bronchiectasis. Of the 12 cases, 11 fell into the group with chronic inflammatory changes and fibrosis without acute suppuration before operation, none was in the group in which minimal changes were found associated with the bronchiectasis, and one was in the group with acute suppuration.

Nine patients had symptoms from residual bronchial stump, 1 of which was following pneumonectomy and the others following lobectomy. The 1 pneumonectomy patient and 5 of the lobectomy patients underwent surgical revision of the stump. Three patients still have stumps from which they continue to have symptoms. Two of these refused operation and in 1 case resection was unsuccessfully attempted. Six of the 8 stumps following lobectomy were in the group in which bronchiectasis was associated before operation with acute suppuration, and the other 2 were in the group that showed chronic inflammatory changes. There was no stump in the group in which minimal inflammatory disease was associated with the original bronchiectasis. The 1 pneumonectomy coming to stump resection had previous chronic inflammatory changes associated with the bronchiectasis. In keeping with these observations, 7 of the 9 residual stumps were resected with tourniquet technique and 2 were divided between clamps. None was handled by individual ligation. This emphasizes the importance of bringing these cases to the operating table with as little suppurative disease as possible so that the surgeon will be able to utilize this type of technique. To wait until acute suppurative changes are present which develop

into the chronic inflammatory type with associated fibrosis will result in higher mortality and higher incidence of complications. Among these are listed the revision of a long bronchial stump as seen in 9 cases of this series.

Sinusitis has been present in 14 cases that were followed postoperatively, and all are lobectomy patients. No patients treated by pneumonectomy were bothered by sinus trouble after operation. Of the 14 patients developing sinus disease, 1 developed positive sputum; 9 had less than ½ ounce of sputum a day; 4 had between 2 and 4 ounces of sputum a day. Of these 9 patients, 1 had an episode of pneumonia five months postoperatively, 1 had lingulectomy postoperatively with continued sputum of 15 c.c. due to sinus disease, and 2 had occasional flare-ups of sinusitis at which time their sputum increased to 100 c.c. and 40 c.c., respectively. In both these cases the tourniquet technique was used. Of the 4 patients having between 2 and 4 ounces of sputum daily, 2 had further bronchiectasis in the middle lobe, and 2 had residual stumps following lobectomy with tourniquet technique for which they refused further resection.

Of the 65 lobectomies followed, 43, or 66.2 per cent of lobectomies, had sputum of ½ ounce or less postoperatively; 10, or 15.4 per cent, had continued sputum daily of 1 ounce and more. Of these 10 cases, 4 had persisting sinus disease, 6 had further bronchiectasis without operation, of which one was followed to her death from bronchiectasis, and 3 had stumps from the initial procedure that continued to give them symptoms. The sputum in the above groups is exclusive of those patients who developed tuberculosis.

Of the 13 cases treated by pneumonectomy none suffered from sinus disease, none had significant sputum postoperatively, and only 1 had minimal sputum (1 teaspoonful).

Symptoms of pain were present following pneumonectomy in 2 cases. In 1 case metastatic carcinoma of the ribs developed; this patient may have had carcinoma of the lung although it was not noted on section at the time the specimen was removed and the primary site was never determined. The second case was complicated by empyema and has not been seen since thoracoplasty. Two lobectomy patients had pain that persisted on follow-up, both following left lower lobectomy. One of these had a residual stump for which she refused further operation.

Four lobectomy patients had dyspnea on exertion with some shift of the mediastinum following operation; in 5 pneumonectomies moderate dyspnea was present postoperatively, and 2 developed scoliosis. Ten pneumonectomies required thoracoplasty procedures, a total of 40 operations being performed on the 13 patients in the pneumonectomy group following resection. Nine lobectomy patients required thoracoplasty, of which 4 were for tuberculosis. Of the remaining 5, 4 had upper lobe lobectomies and the fifth had previously had an upper lobe lobectomy and needed thoracoplasty for the empyema that followed lower lobectomy on the same side.

Ten patients, or 12.8 per cent of the 78 survivals followed for an average of four years, developed tuberculosis postoperatively. Of this number, 2 died of tuberculosis in seven months and sixteen months, respectively. Of the remaining 8 in this group, 5 still have positive sputum; 3 have negative sputum.

In 6 of the 10 cases the tuberculosis developed within a year; in 4 cases the disease was noted after sixteen, twenty-three, twenty-four and thirty months, respectively. In 1 case the disease developed in the pleural space and the patient had never had positive sputum; in all the other cases tubercle bacilli were present in the sputum.

The development of tuberculosis in these 10 cases had no relation to the site of the bronchiectasis. In 1 case the right upper lobe had been removed, in another case, the right upper and middle lobes; in 4 cases the lower and middle or lingula, and in 2 the lower lobe was removed; in 2 cases pneumonectomy had been performed. In 6 cases the tourniquet technique was employed, in 2 cases, individual ligation, and in the 2 remaining cases, division between clamps was the technique used.

The possible mechanism of infection in these cases is worthy of consideration. Undetected tuberculosis may have been present in the lobe removed and spread to the remaining lung tissue at operation. Second, the disease may have been present at the hilum and tuberculous tissue may have been cut across at operation, thus lighting up the previously quiescent disease. Third, the disease may have been lighted up in the remaining lung as a result of overdistention following resection. Last, airborne or contact infection must be considered on a service where it is impossible, owing to overcrowded conditions, to segregate tuberculous patients before the diagnosis has been confirmed.

Of these possibilities, the first is the least likely. If sufficient ulceration were present in the lung removed to cause spread, it should be noted on routine pathologic examination. That tuberculosis may have been present at the hilum is suggested by the high incidence of the development of the disease following tourniquet resection.

In this series of cases, individual ligation was less universally employed than is now the case. Furthermore, the extent of the disease at the hilum required the employment of the tourniquet technique. Of the 2 individual ligations, 1, a pneumonectomy, was the case in which the disease developed in the pleural space following empyema and bronchopleural fistula; in the other, the left lower and lingual lobes were removed together, suggesting division of lung tissue. Dividing the hilum between clamps implies the sectioning of pulmonary tissue and the possibility of cutting across tuberculous tissue. In 1 case sputum became positive after attempted removal of the stump following tourniquet resection, and in another the sputum became first positive, then negative, then positive again after further stump excision eighteen months after the original operation.

Overdistention as a factor in the development of tuberculosis was considered probable in 5 of the 10 cases. In 4 of these the disease developed in the remaining portion of the resected lung. In retrospect, after reviewing the preoperative roentgenograms, it is felt that 2 of these patients may have had unrecognized chronic pulmonary tuberculosis, although only one had a history of previous

tuberculosis. In one case tuberculosis developed on the opposite side after pneumonectomy. In this case enlarged glands were present at the hilum at the time of operation but there was no suggestion made that they were tuberculous; however, sections were not taken at operation.

Of the remaining 5 cases in which tuberculosis developed, 3 had residual stumps following operation. One cleared up with reduction of sputum, a second developed positive sputum after attempted resection of the stump, and the third developed positive sputum after resection, became negative, and then positive again after excision of the stump. In the first of these, resection was by division between clamps, and in the other 2 the tourniquet technique was employed. The fourth was the pneumonectomy case in which tuberculosis developed in the pleural space following bronchopleural fistula, necessitating multiple operations to obliterate the sinus and the pleural cavity. In the fifth case a tourniquet stump repair was done for resection of the left lower and lingula of the left upper lobe which was followed by the development of large amounts of sputum due to tuberculosis of the right side, developing first in the right upper lobe and then spreading to the other side, and to the intestinal tract, resulting in death five months after the disease was diagnosed by the presence of tubercle bacilli in the sputum.

From the analysis of these 10 cases it is evident that several factors could account for the development of tuberculosis following operation for bronchiectasis. All cases should be carefully evaluated by x-ray examination for the possibility of unrecognized tuberculosis before coming to operation. If there is a suggestion of inactive tuberculosis in the remaining lobe following lobectomy or of the opposite lung following pneumonectomy, early thoracoplasty is advisable to prevent overdistention and the possible reactivation of remaining tuberculosis.

These 106 operations for bronchiectasis represent lobectomies and pneumonectomies done during the years 1939 to 1945. Segmental resection in this study was limited to cases in which the lingula of the upper lobe was removed in conjunction with the upper or lower lobe on the left side. In 1 case part of the upper and lower lobes on the right side were removed but the modern type of segmental resection was not employed.

Since then segmental resection has been employed more frequently for bronchiectasis. The principle of the operation is to remove that portion of the lobe in which bronchiectasis is present and preserve as much of the functioning lung tissue as possible. This implies the removal of only the diseased tissue, so that there will not be a lack of functioning lung tissue as the result of pulmonary resection, nor unnecessary removal of normal lung for lack of any easy anatomic cleavage.

The importance of the present study of cases lies in the bearing of the pathology in the resected lung on the postoperative course. The fundamental principles that become obvious in these cases apply to whatever operative technique is employed. Control of the acute suppurative and chronic inflammatory factors in bronchiectasis is as important for a segmental resection as for a lobectomy or pneumonectomy. Suppuration as encountered in this series represents bron-

chiectasis in its most serious form. The advanced stages of suppuration found in some of these patients implied an ignorance of the eventual outcome of the disease by the patient and the doctor.

The mortality in this group, which is so much higher than in the group that presented no acute suppuration, does not represent the same mortality as for uncomplicated bronchiectasis. The surgical risk will be determined by the extent and type of associated suppurative disease.

With the emphasis being laid on segmental resection at the present time it is well to bear in mind that this operation is done with the individual type of technique. It is therefore important to eliminate the suppurative factor as much as possible before bringing the patient to operation. As more experience in this type of technique has been obtained both the mortality and the incidence of complications have been decreased.

In summary, it may be said that resection for bronchiectasis is a less formidable procedure in those cases in which the disease is not associated with acute suppuration as determined by the incidence of complications and mortality following resection. That the incidence of fistula is lower in lobectomy for uncomplicated bronchiectasis than in bronchiectasis associated with suppurative disease focuses the attention on the hilum where fistula occurs postoperatively. When suppurative disease is present, the hilum is involved in the inflammatory process to a greater degree than in simple bronchiectasis where there is no inflammation in the periphery and therefore no involvement of the hilum. Chemotherapy has been of great assistance in reducing inflammation at the hilum, and therefore should be responsible for further lowering the morbidity and mortality of the operation.

As is true of any serious disease, surgery is employed for the purpose of relieving the patient of his symptoms. In bronchiectasis this is ideally performed in the uncomplicated stage of the disease. If the patient has passed the point of ideal operability, we still feel that he ought to be given the benefit of surgery, realizing at the same time that although the risk is greater, the results will be correspondingly more gratifying.

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(See discussion on page 265.)

THE SURGICAL TREATMENT OF BILATERAL BRONCHIECTASIS

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ALTHOUGH the first report of bilateral lobectomy for bilateral bronchiectasis was that of Eloesser in 1933, no report of any considerable series of cases has appeared in the intervening years. In spite of the number of patients available, for one-third of bronchiectatic patients have bilateral disease, and the morbidity and mortality to which they are subject, thoracic surgeons appear to have been cautious in undertaking bilateral resection.

In 1942 Blades and Graham reported 4 bilateral resections with 1 death. In the same paper they mentioned 12 other cases of bilateral bronchiectasis, subjected to unilateral resection, with 3 deaths. As recently as 1947, Bisgard and Swenson reported 4 successful cases of bilateral resection, and from the literature to that date, could collect only 37 further cases, with 5 deaths. Strieder, Overholt, Laird, and others in discussing bronchiectasis have referred to small groups of patients in whom bilateral resections have been done, and there have been a number of individual case reports.

Because conclusions in regard to mortality rate and expectation of cure formed on the basis of reports of small groups of cases from various centers are likely to be misleading, it has seemed worth while to study a group of patients with bilateral bronchiectasis, who have been treated at the Toronto General Hospital and Hospital for Sick Children, Toronto.*

The group consists of 58 patients of whom 27 have had unilateral operations and 31 have had bilateral operations. A total of 94 excision operations have been done on these patients, with 4 deaths, an operation mortality rate of 4 per cent, and a patient mortality rate of 7 per cent. The 4 deaths all followed the second side of a bilateral resection; therefore, in the 58 patients there has been no mortality following operation on the first side, but in the 31 patients who have had excision on the second side there have been a mortality of 13 per cent. It is of interest that these 4 deaths occurred in the first 13 cases. By attention to certain technical details, which will be discussed below, it has been possible to complete the last 18 consecutive bilateral resections without a death.

Forty-eight of the patients in the series were treated at the Toronto General Hospital. In age they varied from 14 to 43 years, with an average of 22 years. The other 10 were treated at the Hospital for Sick Children and varied in age from 4 to 14 years, with an average of 10 years. The 4 deaths all occurred in the older group. The oldest patient on whom bilateral resections have been completed was 28 at the time of the second operation. The younger patients,

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*The patients in the Toronto General Hospital group were operated upon by Dr. N. S. Shenstone, Dr. R. M. Janes, and the author, and in the Hospital for Sick Children group by Dr. R. M. Janes, Dr. F. R. Wilkinson, and the author. The author is grateful to these surgeons for permission to include their cases in the series.

and in particular the children, stand extensive resection better than do the older group, but no definite conclusion has been reached in regard to the age limit for bilateral resection. One patient, 39 years of age, with extremely severe disease involving the right middle and lower lobes and the left lower lobe and lingula has had a resection on the right and will shortly be subjected to operation on the left.

Patients with bilateral bronchiectasis may be divided into two groups on the basis of the degree of involvement of the second side. The first group have severe or moderately severe disease of the first side, with slight or minimal disease of the second side. In the second group the disease on both sides is severe or moderately severe. The policy which has been followed has been to operate only on the more severely diseased side in Group 1, but to do bilateral resections in Group 2.

In this series, 15 patients showed minimal disease on the second side and were treated by unilateral resections. Eleven now have no symptoms, and consider themselves cured; 3 are greatly improved, complain only of slight cough, and have less than 1 ounce of sputum per day. These 14 patients have a satisfactory result from their treatment, but the fifteenth patient demonstrates that the degree as well as the extent of the disease on the second side is important. She has had a resection of the basic divisions and the lingual segment on the left, and a bronchogram shows that there is no residual disease on that side. On the right the disease is confined to the anterior basic division, but in that bronchus is rather severe. The patient still has 3 ounces of purulent sputum per day; she will require a resection of the involved segment.

The second group of patients, 43 in number, have had severe or moderately severe disease in both lungs. Very gross disease has been considered an indication for operation, rather than a contraindication, provided that the upper lobes with the exception of the lingual segment were healthy. These patients have all been improved by the first operation; sputum has usually been reduced by one-third or one-half, the patients have gained weight and had considerable improvement in their sense of well-being. At times, it is tempting to both patient and surgeon to accept a degree of improvement and avoid the risk of a second operation, but as these patients are followed, some show relapse, with increase in their symptoms, and all continue to be subject to the disability and dangers of residual bronchiectasis. For that reason, before any surgical treatment is undertaken, these patients agree to accept the hazards of bilateral resection.

In 31 of these 43 patients, bilateral resections have been completed and 4 others are awaiting operation on the second side. The other 8 patients have had unilateral resection. One patient moved to a distant part of the country after the first operation and was lost to the follow-up, 2 patients are so much improved after the first operation that they are satisfied with their result and have carried on for seven and five years, respectively, 3 patients have refused the second operation in spite of fairly severe persisting symptoms, and 2 of the earliest cases, on more complete bronchographic examination of the second side showed disease too extensive for resection. They should not have been operated

upon, and under the present rigid rule that no operation will be done until all segments of both lungs have been clearly demonstrated by bronchography, they would not have been accepted for surgical treatment.

It is the study of the 31 patients who have had bilateral resections which illustrates the problems in the complete surgical treatment of this disease. Fig. 1 illustrates the resections which have been done in these patients and indicates the types of resection, which resulted in death. The first fatality, in 1940, was seventy days after the second operation from a combination of persisting atelectasis and empyema on the side of operation. With the modern treatment of

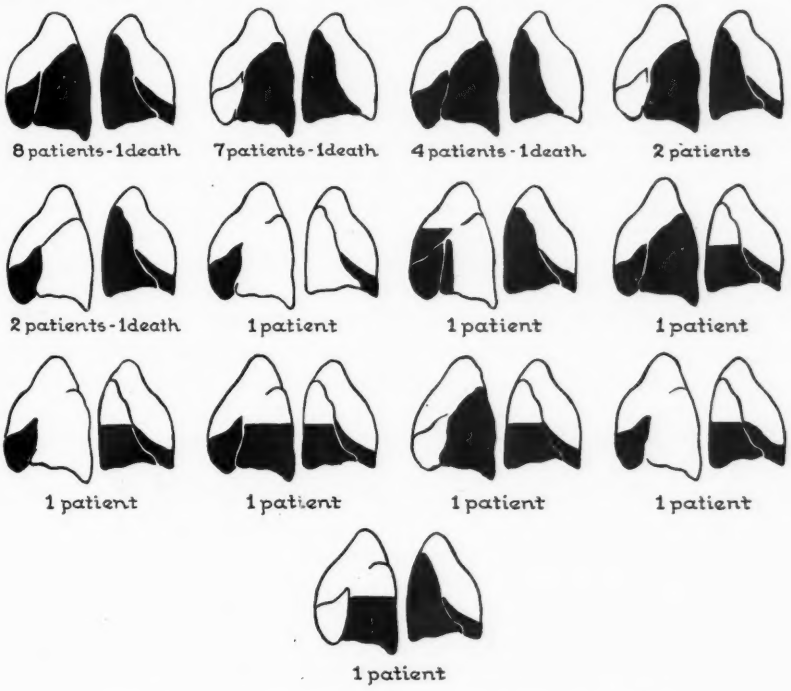

Fig. 1.—A diagrammatic representation of the 31 bilateral resections. The black areas indicate the lobes or segments which were resected.

atelectasis by bronchoscopic suction and the use of penicillin, this death would not have occurred. The second death, in 1942, was twenty-two days after the second resection, from a brain abscess, confirmed at autopsy. Penicillin might have prevented this. The other two deaths, in 1946, emphasize the fundamental problem in this type of surgery. One was twenty-eight hours and the other fifteen hours after the second resection and both were from cerebral anoxia which had been allowed to persist during the operation.

With dissection technique, and the use of antibiotics, sepsis, although still a problem is no longer lethal. Danger to life is due mainly to one factor—cerebral anoxia. During any thoracotomy, respiratory function depends almost entirely on the contralateral lung. If that lung is diseased, and more particularly if it has been reduced to a single lobe by a previous resection, the margin of safety is obviously very narrow. Experience has shown that with proper anesthetic technique a contralateral lung consisting of a single lobe can carry a patient through an arduous resection on the second side provided the airway to that lobe remains clear. If, however, there is any obstruction to the contralateral bronchus, the patient is immediately in grave danger from cerebral anoxia. It is to be noted that this danger extends into the postoperative period to the time when the remaining lung of the side of recent operation has been expanded and taken on adequate function.

In the prevention of anoxia there are two general considerations—the first to develop maximum respiratory function before operation and the second to protect that function by preventing bronchial obstruction. The first consideration affects the choice of side for the first operation, the timing of the operations, the use of breathing exercises to develop function, and in particular to recover it after the first operation; the second consideration implies certain measures before, during, and after operation to reduce the amount of sputum and to ensure that it is evacuated rapidly from the bronchial tree.

Because, as pointed out previously, the danger of anoxia is greater during the second resection because of the reduction of the contralateral lung by the previous lobectomy, in those cases in which there is any appreciable difference in the extent of the disease on the two sides, it has seemed reasonable to do the lesser resection first, so that, at the critical second operation, there will be a larger functioning lung. This policy has been followed in the latter half of the series.

Before undertaking the second resection, time must elapse to allow maximum functional recovery of the first side, and also to take full advantage of the improvement in general condition of the patient. Six months appears to be the minimum interval; more commonly in this group the interval has been nine months to one year. The importance of breathing exercises in recovering and increasing function cannot be overemphasized.

Sinus disease is cleared up as far as possible before undertaking any pulmonary surgery. Before each operation every method is used to reduce and evacuate bronchial secretions. The patients sleep with the foot of the bed elevated. Supervised, effective postural drainage is carried out three times a day. Expectorants are used if the secretions are difficult to raise. Penicillin is usually given by intramuscular injection for two days, immediately before operation. Inhalation of nebulized penicillin or streptomycin, or both, has not seemed to have any special advantage. Finally, thorough postural drainage is carried out immediately before giving the preoperative sedative.

Bronchoscopic suction is occasionally done immediately before and is invariably done at the termination of the operation. All operations are performed under intratracheal ether-oxygen anesthesia with endobronchial suction as in-

dicated. For the past two years patients with bilateral bronchiectasis have been operated upon in the prone position, and this decreases, but does not completely obviate the danger of a spill of mucus into the contralateral bronchus.

Since the two fatal cases of cerebral anoxia in 1946, continuous observations of the arterial oxygen saturation have been made during the operation, in all cases, by means of an oximeter. It has been demonstrated that, provided the contralateral bronchi can be kept clear by suction, the arterial oxygen saturation can be restored and maintained by the frequent application of positive pressure. The greatest value of the instrument is that it indicates when these measures are not effective, and gives warning of the existence of a persisting degree of anoxia which is dangerous. On two occasions the operation has been abandoned after freeing the lobe, because of inability to correct anoxia, and in each case the operation was completed satisfactorily at a later date. It is believed that, without the warning given by the oximeter, these 2 cases might have resulted in fatal cerebral anoxia.

Fig. 2 illustrates the record obtained in a patient with very gross bilateral disease during the resection of the first side. In spite of the prone position, as the lung was freed there was a very heavy spill of secretions into the trachea with resulting acute anoxia. The anesthetist was successful in clearing the obstruction by suction and following that the oxygen level shows the usual intermittent depressions and peaks due to suction and positive pressure.

Fig. 3 illustrates one of the cases in which the operation was abandoned because of inability to clear the opposite bronchi by endobronchial suction.

In the postoperative period every effort is made to keep the bronchi clear of mucus and to obtain rapid expansion of the remaining lung on the operative side. The foot of the bed is kept elevated, the patient turned every hour during the day, every second hour during the night, and persuaded to cough after the judicious use of sedatives. Expectorants are used. Atelectasis of either side is watched for constantly, and if it occurs is treated by immediate bronchoscopic suction. Closed drainage is used in all cases for two to four days, supplemented by pleural aspirations as indicated.

In this period the margin of safety in the respiratory function is exceedingly narrow. Success depends on constant supervision by well-trained nursing and resident staff who can recognize trouble and act quickly. Unless this supervision is available these patients should not be operated upon.

It is by the application of all the measures described above that it has been possible to complete the last 18 bilateral resections without a death.

In this group of patients the result of treatment must be assessed in relation to relief of symptoms and in terms of respiratory function following treatment. In regard to the former, an assessment should not be made until one year has elapsed after the second operation, because in some patients there is residual cough which slowly improves, while in others, after initial improvement there may be some degree of relapse. Of the 31 patients, 27 have survived. One, when last seen, a few months after the second operation, was well, but since then has been lost to the follow-up. Eight patients have been operated upon too recently for final assessment. Of the remaining 18, 15, or 83 per cent, have no

cough or sputum, have no abnormal signs in the chest, and are not subject to more than the average number of colds or chest infections. The other 3 patients are much improved but still have cough and a small amount of sputum. In all these patients the improvement in general condition and decrease in morbidity is striking; they are an exceedingly grateful group.

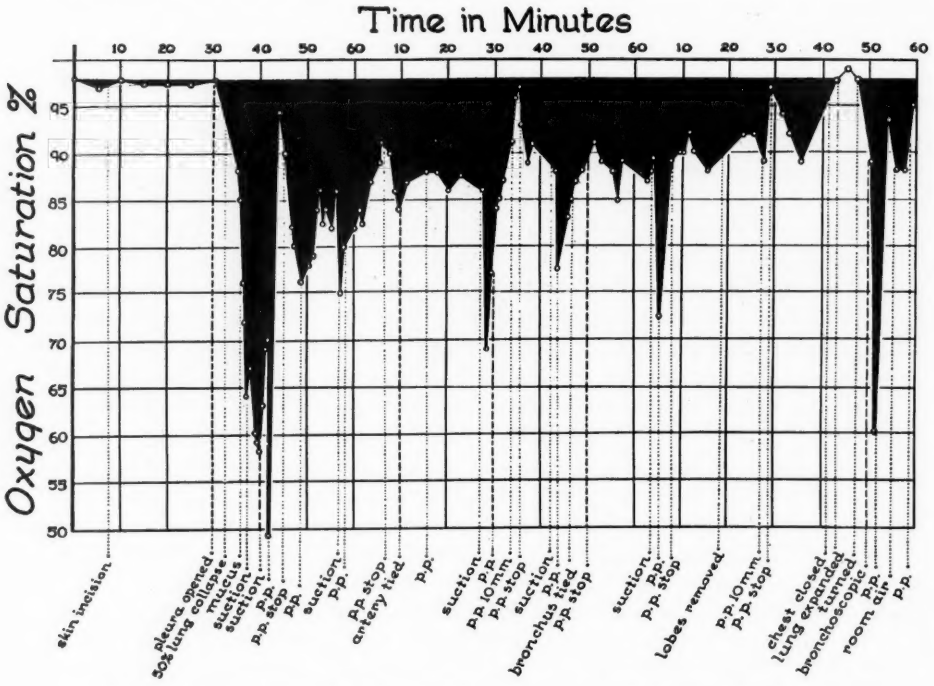

Fig. 2.—Oximeter record of first operation on D. S., 23 years of age, severe bilateral disease, right middle lobectomy with resection of basic segments of lower lobe in prone position. Upper line indicates normal arterial oxygen saturation, the lower line the oximeter readings. The black area indicates the arterial oxygen unsaturation. *P.P.* indicates positive pressure. Note short period of severe anoxia due to obstruction of the trachea by mucus, after the lung was freed. This was overcome by repeated suction and positive pressure.

A remarkable thing about these patients is that none has volunteered a complaint of shortness of breath, although most will admit to shortness of breath on exertion. None is unemployable because of respiratory disability. One man, who has only his upper lobes, without the lingula, has gained forty pounds and is doing fairly heavy work as a steel worker. Another man is a crane operator. The others are all doing light work, chiefly as office workers. The first patient in the series, now aged 35 years, who between 1934 and 1936 had tourniquet resections of the right lower, left lower, and right middle lobes in three stages, each followed by empyema and fistula, has earned her living since as an office worker, has no cough or sputum, states that she is not conscious of any disability, and last year survived pneumonia. Another patient, a woman aged 21 years, who has her upper lobes only, developed severe hyperthyroidism with a basal metabolic rate of plus 84 per cent one year after the second resection. Even with

this increased metabolic rate she showed no unusual shortness of breath. She was prepared with propylthyrouracil, operated on in the usual way, and made a good recovery. These clinical observations appear to support the broncho-spirometric observations of Kay, Meade, and Hughes which demonstrated that, provided chest wall and diaphragmatic function is good, the oxygen absorption following lobectomy may be essentially normal. The place of breathing exercises in regaining this good function is again stressed.

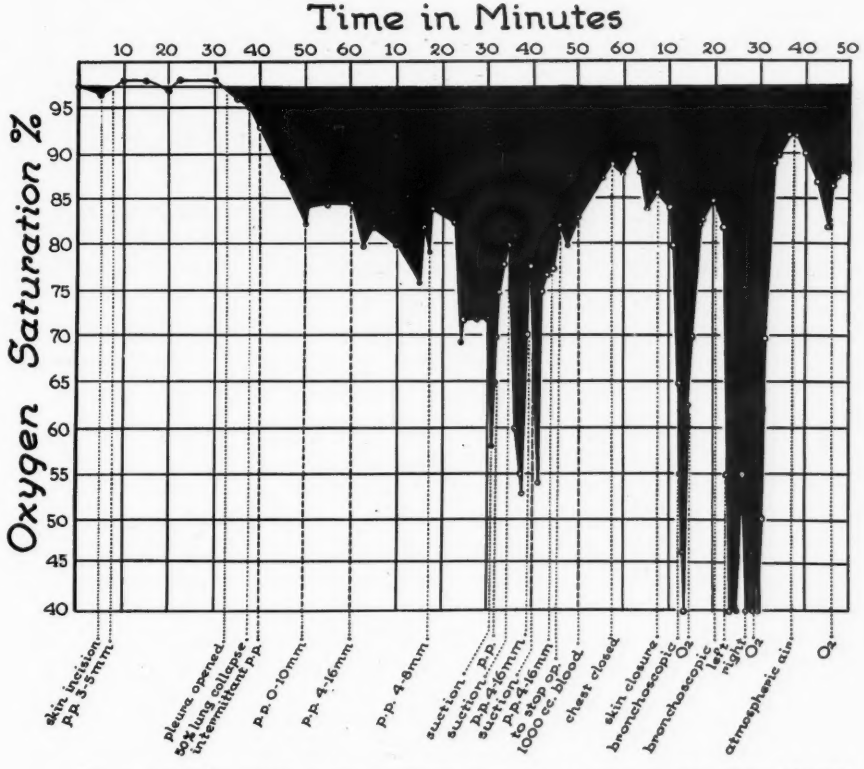

Fig. 3.—Oximeter record of first operation on R. C., 16 years of age, severe bilateral disease, resection of right lower lobe in lateral position. Repeated suction followed by positive pressure was unsuccessful in correcting anoxia. The operation was abandoned and the chest closed. The resection was successfully completed twelve days later.

Although function after these extensive resections is remarkably good, it is natural to attempt to save all healthy lung. On 13 occasions a partial lower lobectomy was done; in 11 of these all the basic segments were resected with preservation of an apparently healthy superior segment. Eight of these resections involved the left lower lobe and were combined with a resection of the lingual segments; the other 3 involved the right lower lobe and 2 of them were combined with a resection of the middle lobe. These 11 operations were performed on 10 patients, 1 girl having had bilateral segmental resection of the basic divisions.

After the 11 operations there were only 5 good recoveries. In the other 6 there were 2 empyemas, 1 persisting fistula without empyema, 2 clotted hemathoraces, and 1 case in which there was incomplete expansion of the superior segment, in spite of repeated bronchoscopic suction and pleural aspiration. Four of these 6 patients have developed bronchiectasis in the retained dorsal segments; 1 has already been resected and the other 3 will probably require resection. These 4 patients are included in the group of patients who have been treated too recently for final assessment.

Because of the high incidence of pleural complications and the tendency to develop bronchiectasis, segmental resection of all the basic divisions with preservation of the superior segment alone has been abandoned, although resection of individual basic segments is still practiced.

SUMMARY

Fifty-eight patients with bilateral bronchiectasis have been treated surgically, 27 by unilateral resection and 31 by bilateral resections. Ninety-four excision operations have been done on the group with 4 deaths, all of which occurred after an operation on the second side.

Fifteen of the patients showed marked disproportion in the distribution of the disease, in that on the second side the disease was slight. These have been treated by unilateral resection, with satisfactory result in all but 1 patient.

The remaining 43 patients showed disease which was severe or moderately severe on both sides. Within certain limitations staged bilateral resection is recommended for this group and has been carried out in 31 patients.

The hazards associated with surgical treatment have been discussed. It has been pointed out that of these cerebral anoxia is the most important, and that it is more likely to occur during or immediately after operation on the second side. Methods which have been used to prevent anoxia have been described, including the use of an oximeter during operation to provide an accurate method of following fluctuations in the arterial oxygen level.

In 18 of the 27 patients who have survived bilateral resection, the final result can be assessed. Fifteen are symptom free and all of them show remarkably little evidence of decreased respiratory function.

The practice of segmental resection of the basic divisions of the lower lobes, with preservation of the superior segment, has been abandoned because of the high incidence of pleural complications and a tendency to the development of bronchiectasis in the previously healthy superior segment.

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DISCUSSION ON "THE RESULTS OF SURGERY IN BRONCHIECTASIS" BY DR. ADRIAN LAMBERT,* AND "THE SURGICAL TREATMENT OF BILATERAL BRONCHIECTASIS" BY DR. FREDERICK G. KERGIN

DR. WILLIAM E. ADAMS, Chicago.—My remarks have to do chiefly with the management of bilateral bronchiectasis. I think Dr. Kergin has given us an excellent presentation of the problem involved. I heartily agree with the principles that he has emphasized that must be observed in management of these patients. A number of years ago Dr. Graham, in reporting on an extensive bilateral resection for bronchiectasis, brought up the question with how little lung function is life compatible. I think in his case he had resected both lower lobes, the middle and the lingula of the left upper. Since that time considerable has been learned regarding the management of suppurative diseases of the lung.

I would like to review briefly our experience with these bilateral cases. (Slides.) This first case was a woman, 28 years of age, who had had bilateral bronchiectasis for a number of years. This was before segmental resection was practiced, and in her case we resected both lower lobes, the right middle, and the liagula of the left upper. The next slide shows a microscopic section through one of the lobes involved and I might point out that making these large microscopic sections routinely has brought out evidence of tuberculosis in a number of cases which would otherwise have been overlooked in the smaller sections.

The second case was a man in his early 30's who, as you can see, has bronchiectasis of the lower lobes, the right middle and the lingula, all of which are completely collapsed; in addition, the lower part of both upper lobes is remarkably emphysematous. This patient represents the most extensive resection we have carried out; it included both lower lobes, the lingula, the right middle and a portion of the upper lobes. This patient had a vital capacity of around 3,700 c.c. in spite of the fact that he had only the upper two lobes functioning. Following operation roentgenographic appearance of the lung was about the same as before with the exception that the diaphragms were somewhat elevated; less dyspnea was experienced than before operation.

Here is the microscopic section of one of the lobes, which illustrates the type of disease present.

In reviewing our experience with these cases, there were 80 in which operation was done for bronchiectasis, 32 of the 66 being bilateral. Of the 32, in 14 the resection was completed and, unlike Dr. Kergin's experience, our main difficulty was following the first stage. The two operative deaths occurred in 2 of these bilateral cases following the first operation. There were no deaths following the second operation. Of these 14, 10 are on full activity, a number are earning their living working a full day. Of the 14 there were 8 women, and 5 have gone through normal pregnancies. This is evidence that these patients are not necessarily pulmonary cripples following bilateral resection. In view of the fact that at the present time we can use segmental resection in selected cases, we are able to do them a great deal of good.

DR. JOSEPH GORDON, Ray Brook, N. Y.—My discussion is rather a departure from the things that have already been said; I should like to pass on to you some information which is perhaps generally known but which has been helpful to me because of its presentation in familiar graphic form. We all know that the pulmonary arterial tree parallels the bronchial tree very closely, as has been shown many times. So much is this so, that even

*See page 246 for article by Lambert.

in disease processes which involve the lung it usually involves the bronchial tree and the pulmonary arterial system as well. The notable exception of this, however, is in bronchiectasis, and these illustrations I am about to show have helped me to understand the pathologic findings and the disturbance of the pathologic physiology in cases with bronchiectasis. (Slides.)

In this slide you see the familiar pattern of a normal bronchogram; the illustration on the right is an arteriogram showing a normal pattern except in the basal portion where there is evidence of emphysema. The pattern of the pulmonary arteriogram is similar to that seen in the bronchogram. During the past seven or eight years we have undertaken at Ray Brook the study of both surgical and post-mortem material by this method of contrast roentgenography. Keeping these two normal patterns in mind the next slide shows an arteriogram with most of the smaller vessels gone. On the right there is marked obliteration of the arterial tree in the presence of extensive disease and is even more so with thoracoplasty on the left. In the lower left corner you see the small divisions of the arterial tree showing a relatively normal pattern, contrasted with marked obliteration of the remaining portion of the tree and in contrast with the previous slide.

In the next slide there is evidence of the usual dilated bronchi in the bronchogram shown on the left in the presence of the atelectatic lower lobe; on the extreme right the contrast medium is in the pulmonary artery. In spite of extensive bronchiectasis because of bronchial involvement, there is a normal pulmonary arterial pattern. The middle picture shows the superimposed bronchogram in the presence of the arteriogram; there is extensive bronchial involvement in the bronchogram, with no evidence of the third and fourth order of the bronchial tree. But in the arteriogram the pattern is normal, showing all the small arterial divisions. In the next slide you see again a similar contrast. This is an excised bronchiectatic lobe, a postexcisional bronchogram on the right showing very extensive bronchiectasis. In contrast to this, however, the arteriogram in the region of the disease is normal. This explains to me the reason for the disparity between anoxia in the patient with bronchiectasis as contrasted with the patient who has more marked pulmonary tuberculosis, and still shows no evidence of oxygen desaturation. Under exercise, as Dr. Cournand pointed out yesterday, the perfusion of the lung increases to a great extent. Here is a lung that is being perfused because it has a normal pulmonary arterial system in the presence of a lung that is so damaged that it cannot be oxygenated. Under exercise the patients become more markedly desaturated as contrasted to the tuberculous patients. This explains to me also the urgency of doing operation on young patients because they are chronically anoxic, when if allowed to go on it leads to some of the difficulties of operation later in life. There is chronic anoxia, failure of development, cyanosis, clubbing of fingers, and hemoptyses.

DR. R. M. JANES, Toronto, Ontario.—I would like to say that I believe the concept which Dr. Kergin has presented with regard to management of bilateral bronchiectasis is sound, and to call your attention to the fact that in the last three years there has been no mortality. I believe his suggestion, and it was his originally, that the lesser side in some cases should be operated upon first, is also sound, in that it leaves a sufficient lung for adequate ventilation during the second stage, and anoxia during and following operation, certainly in our experience in recent years, is the greatest hazard the patient faces.

I should like particularly to call attention to the type of bronchogram developed by our department of radiology. I do not know how generally spot films are being used; spot films similar to those being used in gastrointestinal roentgenology. (Slides.) This is an ordinary posteroanterior and lateral film of the bronchogram of a normal lung. These are additional spot films, which we now employ in all cases; they are taken when it is observed under the screen that the segments it is desired to outline are filled. I think if you had time to study those you would see that they enable you to visualize every individual segment of the bronchial tree.

These have been a great comfort to us and have enabled us to approach operation upon these patients with complete certainty that we knew the total extent of the disease.

DR. WILLIAM LEES, Ann Arbor.—I should like to interject two main points, one with regard to the use of antibiotics in preoperative preparation of these patients. In Ann Arbor for a number of years we have found that the aerosol or nebulization of penicillin solution has, as Dr. Kergin mentioned, not been of too much value in preoperative reduction of foul sputum. In contrast we have used intrabronchial instillation of a solution of penicillin and have found this to be of great value. This was brought to mind by the reports in 1945 and 1946 that penicillin solution in vitro was somewhat more effective against certain organisms than it was in vivo and the bronchi in bronchiectasis are akin to test tubes. To date we have continued to use this as the method of choice. More recently we have used crystallized penicillin or the inhalation of finely divided crystals. I would like to give a word of caution here inasmuch as two patients that I have observed, and three others reported to me by the dermatologists at the University Hospital, have suffered severe ulcerative stomatitis and, therefore, I think we should be careful in recommending utilization of the micro-crystals of penicillin as a routine.

The second point I would like to mention is something which occurred to us about one year ago; that is, the difficulty which all thoracic surgeons have with the upper lobe of the "down lung" side. In the use of the lateral decubitus position with the patient placed in the Trendelenburg position, the upper lobe is the usual catch basin for all secretions, whether it be a tuberculous or nontuberculous lung. It occurred to us that perhaps we should use the reverse Trendelenburg position or, if you will, Fowler's position, inasmuch as we all know the inherent difficulty in attempting to place even a vertebrated suction tip into an upper lobe bronchus for bronchoscopic aspiration of secretions. However, even if the secretions do flow into the other side in the reverse Trendelenburg position, they will be caught by the lower lobe and one can then insert the suction tip into the individual secondary branch bronchi. This has been done in only six cases to date, and may prove to be better than the Trendelenburg position.

DR. D. H. WATERMAN, Knoxville, Tenn.—There are two or three items that I believe deserve some comment; one is the use of spot films mentioned by Dr. Janes. I heartily concur, but would also like to point out the tremendous advantage of posteroanterior stereobronchograms. Dr. Duane Carr, I believe, has advocated this technique for some time.

Our experience with aerosol administration of penicillin has apparently been better than that reported by other clinicians. In bronchiectatic patients we have usually combined it preoperatively with intramuscular penicillin, so possibly the true benefit is difficult to evaluate.

The third point I should like to mention regards the occasional patient who develops additional bronchiectasis following adequate excision of all demonstrably involved tissue. Very little has been written on the subject, yet occasional cases are encountered. We have had 5 in our series. Two have been diabetics, 1 of whom was a situs inversus who had, we felt, a congenital bronchiectasis. In his case we had done a left lower lobectomy, which would have corresponded, of course, to a right lower. He later developed definite bronchiectasis in the left middle and upper lobes and required excision of the remaining portion of that lung. We had another boy with right lower lobe saccular bronchiectasis with an entirely normal bronchogram of the right middle and right upper, who after right lower lobectomy developed severe bronchiectasis in the right middle lobe and the anterior inferior division of the right upper necessitating further excision. We have had 2 others in whom the left lower lobe and lingula were removed, and who subsequently developed bronchiectasis contralaterally in the right middle lobe. In another in whom the left lower lobe was removed leaving an apparently normal lingula, later lingulectomy was necessary.

In discussing the etiological factors that may be back of some of these recurrent bronchiectases, I believe we have to take into account that there may be some individuals, such as diabetics, who quite possibly have a predilection to the formation of bronchiectasis.

Associated sinus disease that has not been thoroughly controlled certainly may play a part in later development of bronchial damage. Chronic bronchitis, which always accompanies bronchiectasis, and which is not adequately treated following lobectomy may also conceivably be an etiological factor. Associated supparative disease with its attendant difficulties, as mentioned by Dr. Lambert, may occasionally be to blame. In postoperative complications I would like to call attention to two or three things of possible etiological significance. One is the fixation of the chest wall that frequently results after lobectomy and that may be largely avoided by going in intercostally rather than by resecting a rib. In this regard, we feel that the restoration of normal chest wall function is very important following resection. Another causative factor may be the fixation and elevation of the diaphragm that often follows lower lobe lobectomy, and another may be the displacement of the remaining lobes with consequent kinking of the bronchi. Overdistention of the remaining lobes might also be a factor—we have seen bronchial dilatation occur subsequently in these overdistended emphysematous lobes. Dr. Kergin pointed out some of the measures that suggest themselves in correcting these etiological factors; certainly postoperative breathing exercises are of real importance.

We feel that this particular group I have mentioned is one that deserves our careful attention. In spite of the excellent results we are obtaining with lobectomy in bronchiectasis we must recognize that there are occasional patients who will develop further bronchiectatic changes.

DR. DERYL HART, Durham, N. C.—In repetition of something said before this Society last year, I would like to mention in the matter of control of secretions a factor which aids in oxygenation during operation and may tend to limit the spread. This is some work done by Dr. Moody on my staff in developing a method of occluding the bronchus. When he wanted to work on this problem I suggested that if he could make something to fit into the bronchus that was of the nature of a cat's-paw, to protrude small claws when he wanted them protruded and could retract them when he wanted to withdraw it without danger of scratching or irritating the trachea or bronchus, that he might be able to keep it in place. This has proved very effective in controlling the secretions from the lobe, it stays in position very satisfactorily except in the presence of extensive granulations where, in making the teeth short enough so they will not protrude through the bronchus, they may not be deep enough to take hold of very extensive granulation tissue. He can insert this into the bronchus of the lobe to be resected, air can be aspirated giving atelectasis of the lobe if it is not already totally atelectatic, and during the manipulation of freeing the lobe it prevents secretions from getting into the bronchial tree and trachea, thereby simplifying greatly the problem of the anesthetist in keeping the airways open. After the lobe is freed and just before the bronchus is clamped, the balloon is collapsed, the teeth retract, and in the same second that this takes place the bronchus is collapsed so that if secretions had not been aspirated continuously during the manipulation, that are retained in the bronchus or caught by squeezing the bronchus with the finger, the instrument is withdrawn.

I simply present this as a method of aiding in keeping the trachea free during operation and of preventing the spread of secretions to other parts of the lung.

COL. JAMES H. FORSEE, Denver.—The recording of large series of patients with bronchiectasis treated by surgical means is significant because the splendid results from surgical management are not yet thoroughly appreciated. We would like to record the experience at Fitzsimons Hospital during the past three years in the management of unilateral bronchiectasis by lobectomy in a series of 164 patients with one death. The mortality figures, however, are not alone the important figures. In the military service, in particular, we have the problem of returning patients to service. Some few years ago it was almost routine practice to retire officers and discharge enlisted personnel who had had a lobectomy for bronchiectasis. During the past eighteen months we have had only one empyema develop

following lobectomy for bronchiectasis, and it is seldom indeed that an officer is retired or an enlisted individual is discharged with a certificate of disability because of the surgical removal of one lobe for bronchiectasis.

I would particularly like to congratulate Dr. Kergin on the splendid presentation of bilateral lobectomies in bilateral disease. We have not been quite as enthusiastic, we will say, in that our number of bilateral lobectomies have been very few.

DR. LAMBERT (closing).—I would like to mention two points in closing; one is that I consider bronchiectasis a progressive disease, not in so far that it involves the bronchi, but in so far that the pulmonary tissue associated with the bronchus will be further damaged. The second point is that in this series I reported there is such a high incidence of complications and mortality that this group represents a group that are extremely ill, and also represent the cases that we did earlier in our experience with resection. Witness the fact that the early cases had almost no individual ligations and we were just beginning to do segmental resections in the later cases. Now, of course, segmental resection is almost universally employed on our service.

I feel that it is important to determine the amount of bronchiectasis present before resection is undertaken. We have not been able in some cases to appreciate exactly whether the bronchiectasis will develop following angulation of a lobe that remains or a segment that remains, or whether it was present originally. I feel that the methods of Dr. Janes and some of the other discussors, particularly the method of Dr. Gordon in the arterial injection, would be of value, although we have not tried that.

This is a very discouraging group of cases and when I summarized them I was amazed at the amount of tuberculosis that developed. I do not know exactly how to account for it. I feel that the mortality was high, but I feel that, considering the type of patient we were dealing with, that the ultimate result would have been fatal had surgery not been employed.

DR. KERGIN.—There is little that I wish to add. I am glad Dr. Adams stressed the remarkable degree of function following these very extensive resections. It seems to us that the degree of function depends not so much on the amount of lung left as upon the excellence of function of the chest wall and the diaphragm. It is because of our desire to protect that function that we have been disturbed about these serious complications following our preservation of the superior segment.

In reply to Dr. Waterman, we have been fortunate and we have not seen fresh bronchiectasis develop on the opposite side in any of this group or in our unilateral cases. We have seen bronchiectasis develop as a complication on the side of operation when there was difficulty with the lung upon which operation had been done, chiefly in maintaining a clear bronchus and in overcoming postoperative atelectasis and infection.

THORACOPLASTY

A Study of Fifty-Six Consecutive Cases

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Los Angeles, Calif.

THORACOPLASTY is today a widely employed surgical procedure to arrest pulmonary tuberculous lesions. We utilize the modern thoracoplasty operation as described in 1925 and 1937 by Alexander,[1] and in 1933 by Carter[2] and by O'Brien.[3] Our aim in performing a thoracoplasty is to bring about a selective collapse of diseased lung with a minimal interference in respiratory physiology.

The cases analyzed in this paper represent a consecutive series of thoracoplasties performed in the Los Angeles Sanatorium in the 33 month period from January, 1945, to September, 1947. This group comprises 56 cases, the latest of which is at least 6 months since its surgery. Our series is a small one, and the cases are comparatively recent. We have tried to analyze the clinical and pathologic factors involved and the early response to surgical collapse.

In the 56 cases, 170 stages of thoracoplasty were performed. Our surgical procedure is the posterolateral approach, as developed by the previously mentioned authors, in which the greater part of three ribs are usually removed at the first stage, and variable parts of two ribs at succeeding stages. Transverse processes are removed below the first rib to obliterate the costovertebral gutter. Anesthesia has generally been local infiltration with Novocain except in the first posterior stage, where nitrous oxide oxygen is given. In a selected group of cases we have found it advantageous to do a preliminary anterior stage. This group comprises the older patients and those with constitutional factors who are expected to respond poorly to the operation. It serves to test a patient's ability to tolerate surgery, especially those who fear the operation. The mild postoperative course of the anterior stage increases the patient's confidence in his ability to withstand the other stages. We have observed no untoward retention of sputum and the results have been generally good.

All of the patients were white. There were 39 men and 17 women. Ages (Fig. 1) ranged from 25 to 61 years. There were 18 per cent between 25 and 30 years, 34 per cent between 31 and 40 years, 35 per cent between 41 and 50 years, and 12.5 per cent between 51 and 61 years. The majority of our cases, 69 per cent, were in the 31 to 50 year age group. Our group is approximately 10 years older than that reported by Aufses[4] and by Meltzer.[5] By classification of disease, 33, or 59 per cent, of our patients had far-advanced disease, and 23, or 41 per cent, had moderately advanced disease (Fig. 2). These are average findings. Meltzer's[5] cases give a higher degree of far-advanced disease, namely 75 per cent. The duration of disease (Fig. 3) we calculate as the time interval

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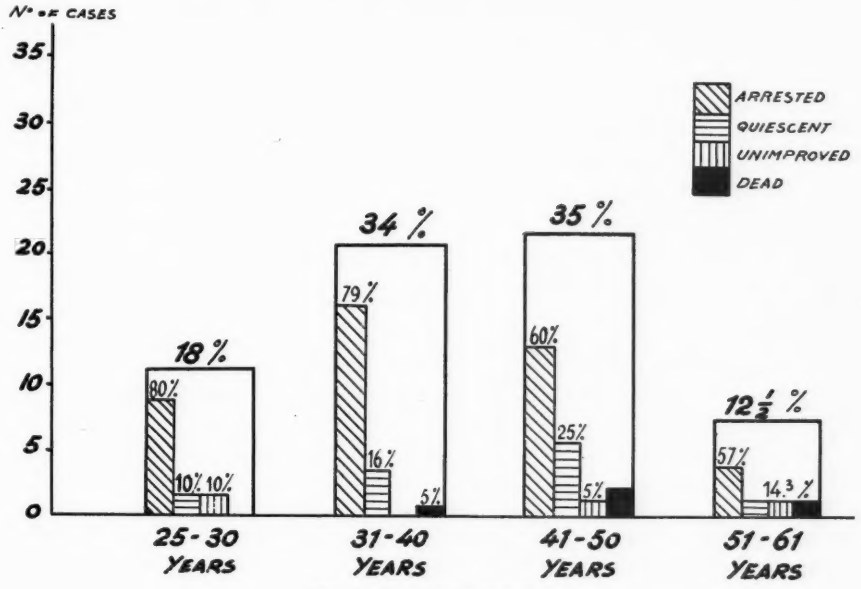

Fig. 1.—Results by age groups.

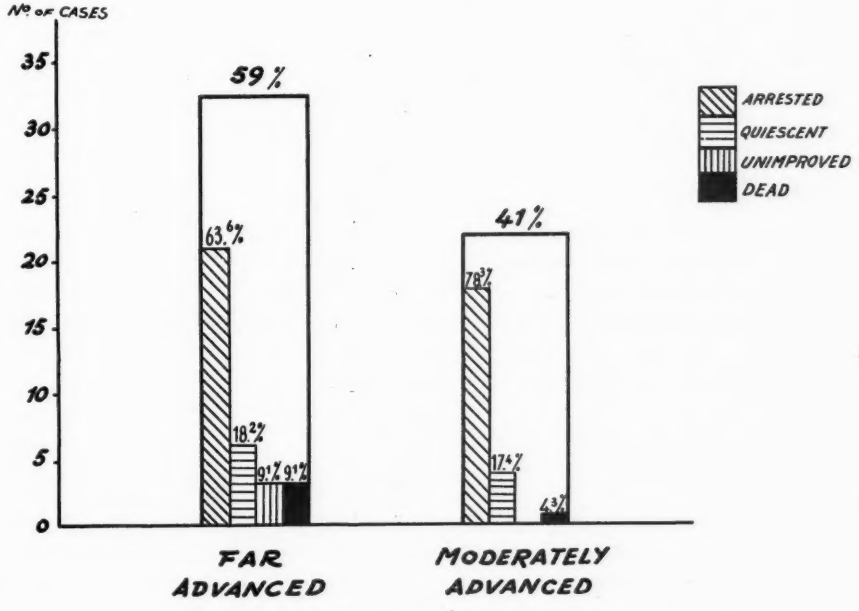

Fig. 2.—Classification of disease result.

between the diagnosis and the surgery. Forty-eight per cent, or 27 cases, had disease from 1 to 5 years, 27 per cent from 6 to 10 years, 12.5 per cent from 11 to 15 years, and 12.5 per cent from 16 to 23 years. Our group shows longer standing disease than is reported by others. Aufses,[4] out of 90 cases, gives 68.8 per cent of his cases between 1 and 5 years, 21.1 per cent between 6 and 10 years, and 10 per cent over 10 years. Meltzer,[5] out of 140 cases, gives 75 per cent having their disease 1 to 5 years, 20 per cent 6 to 10 years, and 5 per cent over 10 years.

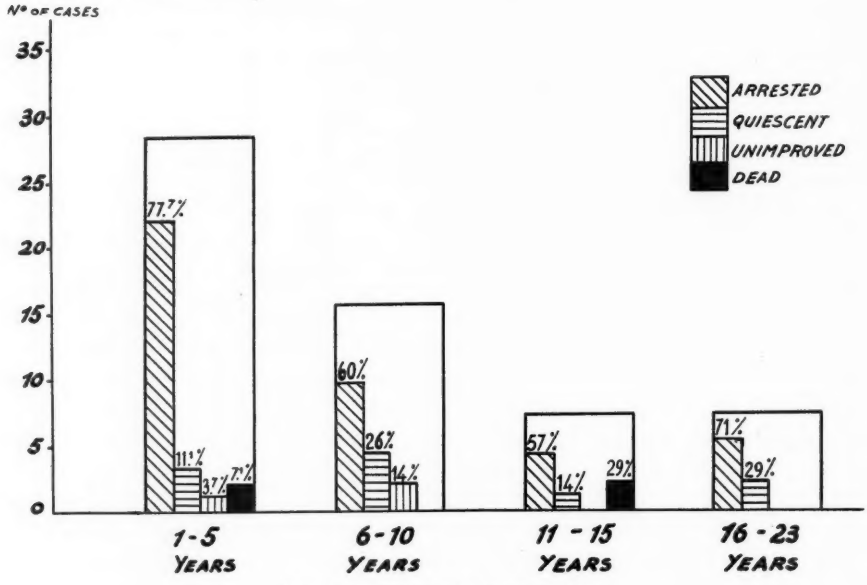

Fig. 3.—Duration of disease results.

Practically all of our patients who become candidates for thoracoplasty have electrocardiographic tracings, among other tests, to determine their cardiac status. We have been interested in finding out how those with unfavorable electrocardiographs fared following thoracoplasty. By unfavorable tracings, we are referring to those showing evidence of myocardial impairment or cor pulmonale. Fourteen such cases were approved for surgery because they appeared to be clinically suited for the operation.

RESULTS

We have tabulated the results according to the standards of the National Tuberculosis Association. By these standards, we submit the following results on 56 cases:

39 cases	70%	Arrested*
10 cases	18%	Quiescent
3 cases	5%	Unimproved
4 cases	7%	Dead

*One patient, after 19 months with negative sputum and a work capacity of 6 hours daily, recently had a positive sputum, the result of a stormy postoperative course after gastrectomy.

In studying the results of thoracoplasty according to age, we found that the groups of patients between 25 and 30 years and between 31 and 40 years gave almost the same results—about 80 per cent arrested (Fig. 1). The group over 40 years showed 60 per cent arrested, which we consider good for this age group. The surgery was borne well by them. Zuelke, Adams, and Bloch[6] reported 68 per cent arrested among 32 patients between 40 and 65 years of age.

Within the group of far-advanced disease, 21 cases or 63.6 per cent were arrested, 6 cases or 18.2 per cent became quiescent, 3 or 9.1 per cent did not improve, and 3 patients died (Fig. 2). Among the moderately advanced disease, 18 cases or 78.3 per cent were arrested, 4 or 17.4 per cent were quiescent, and 1 patient or 4.3 per cent died. Significant we think are the results according to duration of disease (Fig. 3). Best results, as would be expected, are in the group whose disease has been present between 1 and 5 years. This group of 27 cases shows 77.7 per cent arrested. Next best to this group we were surprised to find is the one of long-standing disease, 16 to 23 years. Out of 7 cases, 71 per cent were arrested. What is more remarkable, all of the cases improved. There were no fatalities. While a few cases make percentages difficult to evaluate, we think these findings are unexpectedly good. Our results thus differ from those of Meltzer[5] who found a decline in good results with older disease, especially after 10 years duration. Such a group showing favorable results from surgery after 16 to 23 years of tuberculosis are a naturally select group. They are the patients with "good chronics" who have themselves been able to limit their disease.

We divided the entire series into two age groups (Table I)—those up to and including 40 years of age, and those above 40 years. We found the two groups had almost the same distribution of age of disease. This is a factor that is peculiar to our material. Our sanatorium, being a national institution, draws upon patients from all parts of the country. Many have come here after having been therapeutic failures elsewhere. Some had been refused surgery for various reasons, others had not followed the cure advocated, had refused the operation in the past, and have had a change of heart several years later.

TABLE I. DURATION OF DISEASE AS DISTRIBUTED AMONG YOUNGER AND OLDER PATIENTS

DURATION OF DISEASE	1 TO 5 YEARS	6 TO 10 YEARS	11 TO 15 YEARS	16 TO 23 YEARS
Patients below 41 years of age	14	9	3	3
Patients 41 years and above	13	6	4	4

The 14 patients whose electrocardiographic tracings showed evidence of myocardial impairment were studied for the extent of surgery tolerated, the complications encountered, and the conversion results achieved (Table II). It will be noted that there is no relation between the number of ribs removed and the cardiorespiratory complications found. A total of 40 stages were performed on this group. There were four complications that could be attributed to a weakened heart, one case of cyanosis and dyspnea of three days' duration,

TABLE II. EFFECT OF THORACOPLASTY ON PATIENTS WITH ELECTROCARDIOGRAPHIC EVIDENCE OF MYOCARDIAL DAMAGE

EXTENT OF SURGERY	COMPLICATIONS	RESULTS
3-rib ant. and post. 2 stage	Cyanosis, dyspnea for 3 days following 1st stage	Arrested
4-rib ant. plus Monaldi drainage	No cardiorespiratory complications	Died (CA)
5-rib, 1 ant., 2 post. stages	Dyspnea following 2nd posterior stage	Quiescent
2 post. stages	No cardiorespiratory complications	Arrested
2 post. stages	Limited bronchiogenic spread	Arrested
6-rib, 2 post., 1 ant., revision	No cardiorespiratory complications	Arrested
1 ant., 2 post. stages	Mild decompensation following 3rd stage	Arrested
7-rib, 3 post., 1 ant. stages	No cardiorespiratory complications	Arrested
3 post. stages	No cardiorespiratory complications	Arrested
3 post., 1 ant. revision	No cardiorespiratory complications	Arrested
3 post. stages	No cardiorespiratory complications	Quiescent
3 post., 1 ant. stage	Mild degree shock after 2nd stage	Arrested
3 post. stages	No cardiorespiratory complications	Quiescent
7th rib plus revision	No cardiorespiratory complications	Arrested

another case of dyspnea of a short interval, a case of mild shock, and one of mild decompensation in a person known to have rheumatic heart disease. The only other complication was a bronchiogenic spread. The cardiorespiratory complications are 10 per cent of the 40 operations performed (Table III). We consider this a low figure. None of the complications resulted in a fatal issue, nor were the eventual results of sputum conversion affected by them. Of the 14 cases, 10 or 71.4 per cent were arrested, 3 or 21.5 per cent are quiescent, and 1 patient died of carcinoma. These results are equal to the percentages obtained for the group as a whole. From this study it therefore appears that successful surgery can be completed in cases which are clinically suited for the operation, but where the electrocardiographic tracings seem forbidding.

TABLE III. RESULTS IN THORACOPLASTY ON PATIENTS WITH ELECTROCARDIOGRAPHIC EVIDENCE OF MYOCARDIAL DAMAGE

Total number of patients	14
Total number of stages	40
Number of cardiorespiratory complications attributed to cardiac condition	4
Percentage of operations affected by cardiac complications	10%

Conversion Results		
Total cases	14	
Number arrested	10	(71.4%)
Quiescent	3	(21.5%)
Dead	1	(7.1%)

VITAL CAPACITY LOSS

The cases were analyzed with reference to loss of vital capacity following thoracoplasty. The results are so unexpected that we are presenting them briefly for this reason (Table IV). It will be seen that there is a smaller average loss in the 8 rib thoracoplasty than in the 5 rib, and that one patient with a giant cavity lost 100 c.c. in a 7 rib thoracoplasty, where another lost 1,200 c.c. with the removal of only 3 ribs. We think that vital capacity measurements are unpredictable and should not be made a basis for selecting cases for surgery.

TABLE IV. VITAL CAPACITY LOSS ACCORDING TO EXTENT OF SURGERY

	3 RIB	5 RIB	6 RIB	7 RIB	8 RIB
Extremes of vital capacity loss	200-400	200-1200	200-1200	100-1200	100-800
Average loss	333	666	870	606	533

PRIMARY VS. DELAYED THORACOPLASTIES

Of the 56 cases, 13 were primary thoracoplasties and 41 were delayed thoracoplasties. We define delayed thoracoplasties as those performed following previous reversible collapse measures. Two cases were revisions of original thoracoplasties which had been done elsewhere. As we will see by Table V, primary thoracoplasties give a higher incidence of arrested cases, 77 per cent as against 68.3 per cent for the delayed group. In this matter we agree with Adams and Dufault[7] who advocated thoracoplasty as an elective procedure where it will appear to do the most good without wasting time on measures that do not hold out much hope of success. Everyone recognizes that the majority of patients submitting to thoracoplasty have had one or more "reversible" methods of collapse applied in which the complications were often serious and the end results not so reversible as one might wish. A thickened, rigid pleura, a residual empyema pocket will interfere with satisfactory collapse. Frequently a crushed phrenic nerve fails to regenerate. Eighteen of our patients have thickened pleura, some to a marked degree. Three of these had encapsulated fluid, 12 had obliteration of the costophrenic or cardiophrenic angle from an old effusion that had complicated a pneumothorax; there were 16 paralyzed diaphragms, 11 right and 5 left; in 23 cases the trachea and mediastinum were displaced to the side of disease, in 3 cases to the contralateral side. These influences on the physiopathology of the thoracic cage did not favor a good collapse. It is surprising indeed that the modern thoracoplasty operation does give good results in the face of such obstacles.

TABLE V. A COMPARISON BETWEEN THE RESULTS IN PRIMARY THORACOPLASTIES AND IN THORACOPLASTIES FOLLOWING PREVIOUS REVERSIBLE COLLAPSE MEASURES

	NO. OF CASES	ARRESTED	QUIESCENT	UNIMPROVED	DEAD
Primary thoracoplasties	13	10 (77%)	1 (7.7%)	1 (7.7%)	1 (7.7%)
Delayed thoracoplasties	41	28 (68.3%)	8 (19.5%)	2 (4.9%)	3 (7.4%)
Revisions	2	1 (50%)	1 (50%)		
Total	56	39 (69%)	10 (18%)	3 (5%)	4 (8%)

PATHOLOGY

Twelve patients had unilateral disease (Table VI). Ten of these cases were arrested after surgery and two became quiescent. That is 100 per cent improvement in the unilateral group. Of the 44 patients with bilateral disease, 29 or 66 per cent of the cases were arrested, 8 or 18.2 per cent became quiescent, 3 or 6.8% were unimproved, and 4 or 9% of the patients died.

TABLE VI. RESULTS ACCORDING TO DISTRIBUTION OF DISEASE

	NO. OF CASES	ARRESTED	QUIESCENT	UNIMPROVED	DEAD
Unilateral disease	12	10 (83%)	2 (17%)	—	—
Bilateral disease	44	29 (66%)	8 (18.2%)	3 (6.8%)	4 (9%)
Total	56	39 (69%)	10 (18%)	3 (5%)	4 (8%)

We have attempted to classify our patients by type of pathology to study the effect of thoracoplasty. We realize there are difficulties in any such classification. Few cases present one pathologic picture not complicated by another. We have divided them (Table VII) according to the most important problem in each case. Two-thirds of the first group, fibrotic or atelectatic lobe with cavity, were arrested. This is not remarkable since this type is expected to respond best to surgical collapse. Those with tension cavities and evidence of bronchial disease were all improved. We did not expect such good results. The group in which bronchiectasis was the main feature associated with small cavities also gave favorable results. Of the 4 giant cavities all were converted except in one patient who died of cancer before the surgery could be

TABLE VII. RESULTS ACCORDING TO TYPE OF PATHOLOGY

PATHOLOGY	NO. OF CASES	ARRESTED	QUIESCENT	UNIMPROVED	DEAD
Cavity in fibrotic or atelectatic lobe (no resisting bronchial disease)	22	14	4	1	3
Tension cavity with demonstrable bronchial disease	7	5	2		
Tuberculous bronchiectasis with cavitation	5	4	1		
Giant cavity	4	3			1
Cavitation with atelectasis of entire lung	2	1	1	1	
Caseous pneumonic disease with cavitation	13	10	2		
Pulmonary tuberculosis with mixed empyema	1			1	

TABLE VIII. GIANT CAVITIES

CASE NO.	SIZE OF CAVITY	AMOUNT OF SURGERY	RESULTS
17	5 by 11½ cm., depth from sternum to spine; occupies all right upper lobe	4-rib anterior stage, Monaldi	Dead; bronciogenic CA with metastases
22	5½ by 9 cm., from clavicle to 4th anterior rib	4-rib anterior stage; 7 posterior ribs plus cavernostomy	Converted 6 weeks after last stage; arrested
36	7 by 9 cm., right upper lobe	3-rib anterior, Monaldi; 7-rib posterior	Converted 7 months after last stage; arrested
51	13 by 4½ cm., occupying all left upper lobe	8-rib posterior	Cavity closed with 3rd stage, converted 1 month after 3rd stage; arrested

completed. In two cases of cavity and atelectasis of the whole lung, one was arrested and one became quiescent. One patient with pulmonary tuberculosis and a mixed empyema had conversion of the sputum but is unimproved now since the wound broke down after a complete thoracoplasty. Thirteen cases of caseous pneumonic disease with cavitation are grouped together. The giant cavities occupied all or most of a lobe (Table VIII). Surgery was not completed in the first patient. In the second, closure was with a 4-stage thoracoplasty followed by a cavernostomy. In the third, closure was with the aid of a Monaldi, and the resection of 3 anterior and 7 posterior ribs. In the last case, a cavity occupying the entire lobe, closure and conversion were with an 8-rib posterior thoracoplasty.

We are pleased with our results on giant cavities. Excluding the first case that was lost to us because of a nontuberculous disease, the three remaining cases were converted. Shapiro and Muntz,[8] who had excellent results in their work on old-age patients, showed less favorable results with the giant cavities. Out of 8 cases, one was converted, 4 nonconverted, and 3 patients died. Meltzer[5] reported 60 per cent conversions among the giant cavities in his series.

COMPLICATIONS

Of the 56 patients, 30 cases had no complications, and 25 cases had 34 complications of which 24 were surgical (Table IX). The deep wound infections involved the muscular and subcutaneous tissues. In no case were tubercle bacilli cultured. Staphylococcus was the usual organism grown. There have been no wound infections within the past year of this series. This is in part due to the use of penicillin routinely and also to employment of finer suture material. Five cases developed effusions that required aspiration; some of these were serous and some had variable amounts of blood. Two of these resulted in wound separation with delayed healing. There were two cases of shock. These responded rapidly to intravenous infusions. A transient pneumonitis disappeared in 16 days roentenographically, and may have been an autotuberculinization. A spread showed complete absorption in four months. The pleural tears were small and both were closed at the time of surgery. A moderate amount of subcutaneous emphysema followed one of them. The atelectasis followed the first stage of collapse of a giant cavity of the left upper lobe with spillover into the lower lobe. Bronchoscopy was done on the patient a few days following the first stage and the secretions were aspirated permitting aeration of that lobe. The case of cardiac decompensation was in a young man known to have rheumatic heart disease. He had a blowing systolic murmur on admission and had had an attack of auricular fibrillation before surgery. A right pneumothorax was unsuccessful and a thoracoplasty was planned. He withstood an anterior and two posterior stages well, but three weeks following the second posterior stage he had an acute episode of fibrillation with heart enlargement and some decompensation. For this reason the planned third stage had to be abandoned. This patient has since had negative sputum for the past eight months. The

Horner's syndrome followed an apicolysis. The enophthalmus disappeared in four months. There were four cases of dyspnea, one of which was known to be asthma, the others, unexplained. The tachycardia appeared twice in the same patient. The two mental reactions are included only because we think it significant that some patients react in this way following surgery. This is to be expected since mental instability is frequently encountered in most groups of tuberculous patients. The depressive stage cleared up in a few weeks. The paranoid condition was an exacerbation of a previously diagnosed schizophrenia. This cleared up a few months after surgery.

TABLE IX. COMPLICATIONS FOLLOWING SURGERY

Deep wound infection	6
Superficial wound infection	1
Excessive wound effusion	5
Wound separation	2
Ecchymosis around wound	1
Shock	2
Transient pneumonitis	1
Spread of disease	1
Pleural tear	2
Subcutaneous emphysema	1
Atelectasis	1
Cardiac decompensation	1
Horner's syndrome	1
Asthma	1
Dyspnea (unexplained)	3
Tachycardia (same patient)	2
Reaction to penicillin	1
Reactive depression	1
Paranoia	1
	34

STREPTOMYCIN

Nine patients received streptomycin therapy (Table X), 8 in connection with surgery and one 18 months following an unsuccessful thoracoplasty when tracheobronchial disease was discovered. The good results in the first case listed in Table X can be certainly credited to streptomycin. This patient had developed an acute tuberculous pneumonia on the same side for which surgery was planned. In addition she had a specific laryngitis and tracheobronchitis. She was extremely toxic, registered a temperature of 103° F., and her condition appeared critical. On a course of 2 Gm. of streptomycin daily, she made a remarkable improvement, the symptoms abated, and the lesion became stabilized. A three-stage thoracoplasty was then performed. The patient made an uneventful recovery. The dramatic change brought about in this patient under streptomycin is very impressive. It is highly probable that without streptomycin she would not have survived the fulminating disease. Certainly she would not have been made ready for surgery.

In two cases streptomycin was given as protection during collapse of cavities in patients with low vital capacities. It accomplished its purpose in both of these cases. In one of these conversion lasted for three months following

TABLE X. STREPTOMYCIN

INDICATION	DOSAGE	RESULTS	COMPLICATIONS
Acute pneumonic spread in left upper lobe where thoracoplasty had been planned for large tension cavity; acute tuberculous, laryngitis, and tracheobronchitis	2 Gm. daily, 5 days of the week, for a period of 5 months	Dramatic improvement in toxic symptoms, control of fever, stabilization of lesion, surgery performed, case arrested	Dizziness
Protection for surgery in patient with low vital capacity and bilateral disease	1½ Gm. begun 10 days before surgery & continued for 3 mo.	Temporary conversion of sputum; patient died of anoxia 4 mo. after surgery	Elevation N.P.N., lowered PSP, dizziness
Protection for surgery, tension cavity; low vital capacity; contralateral extrapleural oleothorax	1½ Gm. 5 days weekly, 2½ mo.	No spread of disease, sputum conversion, arrested	Dizziness
Protection in collapse of tension cavity	2 Gm., 1 mo.	Sputum conversion 3 wk. following discontinuance of str.; arrested	Maculopapular rash
Protection for collapse of giant tension cavity	1½ Gm. 5 days weekly, 3½ mo.	No spread of disease; sputum conversion; arrested	None
Bronchiogenic spread after 1st stage	2 Gm. daily, 4 mo.	Arrest of spread, stabilization of lesion; converted; arrested	Rise in N.P.N.
Protection with surgery in a patient who had shown poor resistance to disease; a contralateral spread a few months before surgery	1½ Gm. 5 days weekly, 11 wk.	No spread of disease following surgery, still pos. because of incomplete collapse of cavity; quiescent	Febrile reaction
Protection during surgery (cavernostomy for residual cavity following thoracoplasty)	2 Gm. daily for 11 days	Conversion of sputum for 1st time following cavernostomy; arrested	General urticaria, angioneurotic, edema of eye and forehead
Tracheobronchial disease of left main stem bronchus 18 months following unsuccessful thoracoplasty	1 Gm. daily	Treatment not completed; slight improvement noted	None

surgery. She died one month later, and a post-mortem showed a large cavity in the superior segment of the right lower lobe. The other patient who had a tension cavity and a borderline vital capacity, weathered the surgery without incident, had conversion, and the condition is now arrested. Two other cases had streptomycin for surgery on tension cavities. Both did well and conversion took place. A bronchiogenic spread was controlled in one case (it might have been controlled without the drug). One patient, who showed poor resistance to the disease, was given a course prior to surgery. While no spread developed the sputum was not converted either, but this must be attributed to incomplete surgical collapse of the cavity. One patient received it in connection with a cavernostomy following surgery. The sputum was converted and the disease was arrested.

Seven cases of the 9 showed some form of complication. The dizziness persisted after cessation of treatment for several weeks. None of the complications was permanent.

A study of the time relation between surgery and sputum conversion shows that results in 25 of the 40 cases that had conversion (62 per cent were made negative within one month of the surgery. Six, or 15 per cent, had conversion from one to two months later. Four or 10 per cent from 2 to 4 months, and 5 or 12½ per cent converted between 7 and 13 months. The reasons for nonconversion are listed by cases (Table XI). Bronchiectasis, thickened pleura, and tracheobronchial disease account for some failures. Spread of disease and incomplete surgical collapse for the others.

TABLE XI. REASONS FOR NONCONVERSION OF SPUTUM

CASE NO.	REASON
2	Numerous multilocular cavities below the main cavity, five weeks between each stage.
10	Spread of disease to left apex
14	Extensive thickened pleura, pleural pocket above partially collapsed lung before surgery, tracheobronchial disease
17	Died from CA before collapse could be effected
18	Failure of cavity to collapse, probably due to atelectasis of right upper lung
25	Failure of cavity to close
32	Incomplete collapse of cavity
33	Numerous cavities, bronchiectasis; delay of second stage, due to wound infection
40A	Incomplete collapse following revision
41	Tracheobronchial disease and bronchiectasis; persistent cavitation following collapse
45	Bronchial stenosis; atelectasis of left upper lobe
47	Bronchiectasis; incomplete collapse of cavity
50	Numerous calcifications in region of cavity preventing adequate collapse

DEATHS

There were 4 deaths in all.

The first death (first case of the series) was an anesthetic death.

The second patient died of anoxia a few hours after the third stage of a right thoracoplasty. Autopsy findings revealed no surgical cause of death. Collapsed cavities were found in both apices. The one in the left was collapsed by artificial pneumothorax. There was a thick pleura on the left, with bilateral

pleuritis, and old healed mitral endocarditis. It is felt that the pneumothorax involving the emphysematous left lung caused great strain on the respiratory reserve. Preoperative bronchospirometry might have called our attention to the low respiratory reserve of the contralateral lung in which case surgery would have been withheld.

The third patient died of anoxia 4½ months following surgery. Autopsy revealed bilaterally adherent pleura, emphysema, and residual cavity in the superior segment of the right lower lobe. The cardiorespiratory reserve was too low to survive the surgery.

The fourth case was that of a giant cavity for which an anterior stage and Monaldi drainage were completed. The patient began developing pathologic fractures in the extremities. Further investigation revealed a rapidly enlarging bronchiogenic carcinoma. In the face of all this the surgical collapse of the cavity had to be abandoned.

SUMMARY

We have presented an analysis of 56 consecutive cases of thoracoplasty performed at the Los Angeles Sanatorium in a 33 month period. The material is of interest in that it represents a relatively greater proportion of old patients and those with long-standing diseases. Some decided to have surgical collapse done after refusing for many years.

In review it can be stated:

1. The younger patient has a better chance of having the disease arrested with thoracoplasty than the older one (80 per cent below 41 years of age; 60 per cent above 40 years of age).

2. The extent of disease has a direct bearing on the results of collapse. The worst group, far-advanced disease, gave 63.6 per cent arrested.

3. Where the disease is between one and five years' duration the best results may be expected. Our study shows that the group where disease has been present between 16 and 23 years did next best and the results were better than either the 6 to 10 year or 11 to 15 year groups.

4. Patients with stabilized electrocardiographic evidence of myocardial damage, but who appear to be clinically suitable for surgery, may be expected to tolerate staged thoracoplasties surprisingly well with few complications attributable to the heart condition. The conversion results are as good as for the noncardiac cases.

5. Primary thoracoplasty gives better conversion results than delayed thoracoplasty, 77 per cent against 68.3 per cent, and is to be preferred especially in the middle and older age group. The complications following "reversible" collapse procedures may jeopardize the results of a thoracoplasty.

6. A study of the results of collapse on different types of lesions leads to no conclusive findings in our series. However, the tension cavities were all improved. Of the patients with giant cavities (occupying all or most of the lobe) all whose surgery could be completed had arrested conditions.

7. Most complications were of a minor nature and have had little influence on the final results.

8. Streptomycin has been used with gratifying results. Its greatest usefulness in connection with surgery, it would appear, is to prepare patients with tracheobronchial disease and those whose surgery must be delayed because of a recent spread. In doses of 1 Gm. daily, it is well tolerated. In higher doses complications are frequent.

9. Our results out of 56 cases are 39 cases or 70 per cent arrested, 10 cases or 18 per cent quiescent, 3 cases or 5 per cent unimproved, 4 cases or 7 per cent dead.

Of the four deaths one was of a nontuberculous nature. Of the remaining three, one was an anesthetic death; the other two patients died of anoxia, one, early after surgery, the other, late (4½ months).

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TEMPORARY HIGH POSITION OF THE DIAPHRAGM

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HIGH position of the diaphragm is a condition met with not infrequently. We know of the paralysis of the diaphragm caused by a lesion of the phrenic nerve. Usually this is due to a surgical operation, but neurotrophic toxins, especially diphtheria, may also affect the phrenic nerve. In addition to this, the high position of the diaphragm due to a mechanical cause is known: (1) the diaphragm is pushed upward by a subphrenic abscess or by a large liver (tumor, echinococcus, and the like); (2) the diaphragm is pulled upward by a process of shrinkage in the lung or by atelectasis of the lower lobe, due to bronchostenosis.

During the last few years we saw a dozen patients with high position of the diaphragm due to a quite different cause. It always concerned the right half of the diaphragm; only once was the left half simultaneously affected. The high position was temporary; sometimes after a few days, usually after a few weeks, the situation returned to normal. Our series consists of two groups: (a) patients who had undergone extirpation of the gall bladder and developed high position of the diaphragm a few days later; (b) patients in whom the high position came about after a pulmonary process or pleurisy, situated near the diaphragm.

In both groups we have been able to demonstrate that the high diaphragm had compressed the basal parts of the lung. The pressure of the diaphragm had no effect on the upper lobe.

CASE REPORTS

CASE 1.—In October, 1945, a 41-year-old man was operated upon for cholelithiasis. A few days after the operation he developed a pain in the right side and grew feverish. The limitation between lung and liver seemed to be elevated on the right, which was especially obvious on the photograph. A dome-shaped limitation was seen. A subphrenic abscess was considered, but it was noted that the edge of the liver, which was palpable before, had now disappeared. The wall of the abdomen could easily be pushed into an empty space under the ribs. It must be accepted that the liver had been displaced upward. This emptiness was noticed in other cases. The temperature soon fell and the man went home; the diaphragm remained higher on the right and moved but little. After a few months we saw the patient again: the diaphragm was one fingerbreadth higher on the right than on the left and moved normally.

We saw six additional patients with a high position of the diaphragm after an operation on the gall bladder, who showed the same course. One patient (Case 2) showed the same condition after cholecystitis; she had not been operated upon.

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CASE 2.—A 44-year-old woman, suffering from cholelithiasis, had an attack of biliary colic coupled with high temperature, after eating eels. There was a dullness in the right lower lobe, and suppressed vesicular breathing. During x-ray examination the diaphragm appeared to stand high and move little (Fig. 1). At home she had been given sulfathiazole. This was stopped, and the temperature fell immediately. Six months later she was requested to return for a follow-up examination. Both halves of the diaphragm were equally high and moved normally (Fig. 2).

Cases 3, 4, and 5 are examples of the second group:

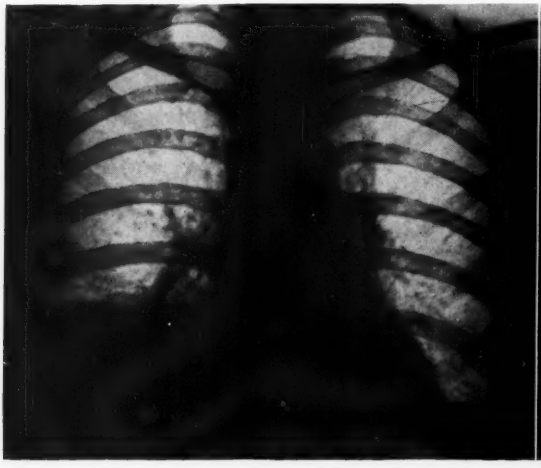


Fig. 1.

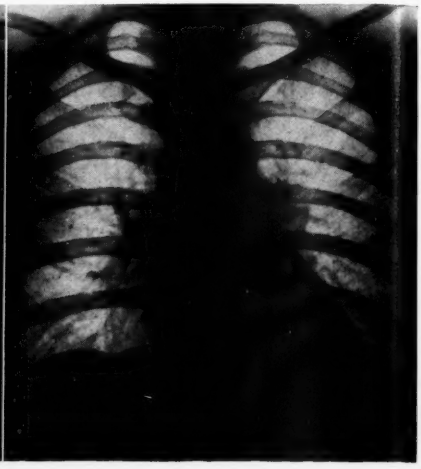


Fig. 2.

Fig. 3.

CASE 3.—A 36-year-old man was sent to us by his general practitioner because of exudative pleurisy. He had dullness and suppressed vesicular breathing above the right lower lobe. The man did not feel ill. He had no fever and the sedimentation rate amounted to 10 mm. On the examination before the roentgen screen, there appeared to be only a very slight quantity of liquid; the dullness was caused by the fact that the diaphragm was a handbreadth too high and did not move.

It will perhaps not be surprising that our thoughts turned to a bronchostenosis, resulting in atelectasis and high position of the diaphragm. Bronchoscopy revealed nothing exceptional, nor did bronchography show any occlusion. It did appear, however, that the lower lobe was far too small. This was especially obvious on the lateral photograph (Fig. 3). The upward displaced diaphragm had compressed the pulmonary tissue around the basal bronchi and the lower part of the middle lobe. The upper lobe, however, was completely normal. Also the apical segment of the lower lobe revealed no changes, except in the lowermost part. With difficulty 20 c.c. of pleural liquid were aspirated and tubercle bacilli were cultivated from it. A strict rest cure was prescribed. After ten weeks neither physical nor x-ray examination revealed any anomalies. The lower lobe had completely re-expanded.

Fig. 4.

CASE 4.—A 27-year-old man had had a cough for a fortnight; he was short of breath and had a pain in the right side. In the right lower lobe there was dullness and suppressed vesicular breathing. Examination before the roentgen screen showed the diaphragm to stand a handbreadth too high and to move but little. Again there was a very small quantity of fluid containing lymphocytes. The cultures remained sterile. Reaction to Pirquet's test was weakly positive. The temperature varied between 92° and 94° F., but was normal after no

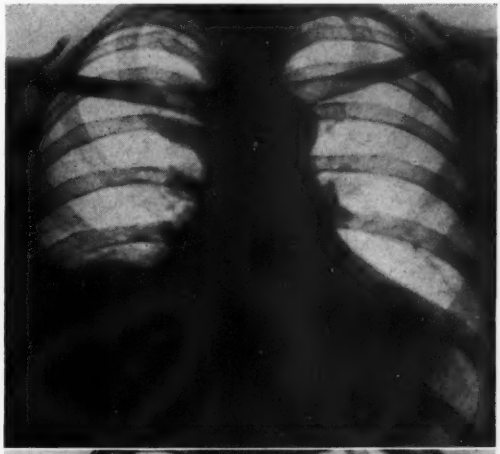
more than three days. Sedimentation rate was 36 mm. Bronchoscopy showed no anomalies. The bronchograms revealed that the pulmonary tissue around the basal bronchi had been compressed, and that there was a filling of only bare bronchi. This is apparent from the lateral x-ray film (Fig. 4). The lower lobe had collapsed (compression-atelectasis), the lower part of the middle lobe was also slightly reduced, but the upper lobe had a normal size and was in its usual place. The man remained in bed, and he rapidly recovered. After six weeks neither physical nor x-ray examination revealed any anomalies. Sedimentation rate was 2 mm. Lipiodol was introduced once more and it appeared that the lower lobe had normally re-expanded, as is visible on the lateral roentgenogram (Fig. 5).

Fig. 5.

CASE 5.—Our advice was asked for a 31-year-old woman, who had been confined three months earlier. On the ninth postpartum day she had suddenly developed a pain in the right side. She had a tight feeling in the chest but did not expectorate blood, nor have a swelling of the leg. However, in view of these conditions, a pulmonary embolus must be considered. The general practitioner soon found a massive dullness. Once more the symptoms were: the diaphragm a handbreadth too high, hardly moving, and in the pleural cavity a very small quantity of fluid. In this fluid *Streptococcus viridans* was found. When bronchography was performed, the upper and middle lobe appeared normal. Of the lower lobe only the first dorsal segment was anatomically normal, the rest of the lower lobe had collapsed, due to the diaphragmatic pressure. Six weeks later the lobe had returned to normal.

The same symptoms were observed by us in one patient who had had pneumonia, and in another who had undergone a rest cure for a tuberculous pleurisy on the left side. Suddenly he developed a pain on the right side and a tight feeling in the chest: he had a high position of the diaphragm. When bronchoscopy was performed (de Graaf) no occlusion of a large bronchus was found After two weeks the situation had cleared.

Fig. 6.



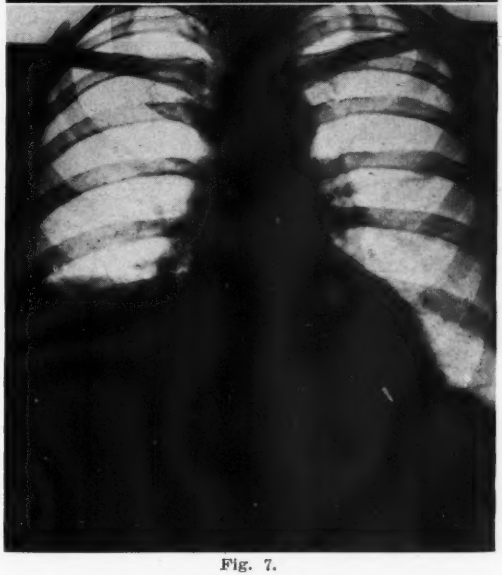


Fig. 7.

We have described patients suffering from a temporary paresis of the diaphragm. This may be caused by a process above or under the diaphragm. What causes the paresis we do not know. It is plausible to think of an inflammatory process of the diaphragm: a diaphragmatitis.

One might think that the collapse of the lower lobe resulted from the fact that the other lobes had been fixed by the pleurisy. We were, however, able to demonstrate that the same lesions are present in the patients with high position of the diaphragm after operations of the gall bladder.

Fig. 8.

CASE 6.—A 35-year-old woman had been operated upon for cholelithiasis. She proved to have a recurrence. At the second operation no stones were found. There were, however, notably many adhesions between liver and stomach and colon, which were loosened. Three days after the operation the patient had a tight feeling in the chest and both halves of the diaphragm were too high (Fig. 6). After a few days the left diaphragm was normal again (Fig. 7), but the right half was still too high. When the woman had sufficiently recovered from the operation, a lipiodol filling was applied on the right side (de Graaf) where once again the largely collapsed right lower lobe was visible. This appears very clearly on the lateral roentgenogram (Fig. 8). It can be noted that the basal bronchi lay closely packed and were crowded toward the back. The middle lobe was not filled.

The diaphragm contains many blood vessels and lymph vessels. It is plausible to think that an inflammatory process in the lower lobe may also attack the diaphragmatic pleura, and that thus the inflammation may reach the diaphragm itself. A similar explanation might be accepted for the cases after operations on the gall bladder—an inflammation of the bed of the gall bladder reaching to the diaphragm. Owing to the operative trauma or the inflammation, fibers of the phrenic nerve may be damaged. Proof for this pathogenesis we do not have; it is a theory, no more. The possibility of a reflex has been proposed, but it is difficult to imagine that a high position of the diaphragm due to a reflex can continue to exist for a period of weeks.

It is a moot point whether a certain change in pressure works on the lung locally (Tendeloo) or spreads equally over the whole lung. This problem is of the utmost importance for pulmonary physiology; for example, is there an equal or an unequal pulmonary ventilation? It is also very important for pulmonary pathology, which in the Netherlands was pointed out especially by Tendeloo (location of pulmonary diseases). In this connection the frequently applied operative collapse therapy should be considered. Should a local or a general effect on the lung be expected from it? From our cases it appears that a lesion of the diaphragm works chiefly locally: the upper and middle lobes remain in their places, whereas the main lower part of the lower lobe and sometimes also the lower part of the middle lobe are compressed. It is clear that the reduction of the lung is achieved all but completely at the expense of the parts of the lung situated near the diaphragm.

We also often found a similar restricted influence on the lung in space filling processes in the thorax, as large tumors, pleural exudate, and thickening. The bronchograms are in this instance often almost the same, showing a compression only of the neighboring parts of the lung.

The bronchi in the compressed parts of the lung show a typical picture. The same was observed in cases of pulmonary compression due to large mediastinal tumors and in affections of the pleura. Only the larger ramifications are filled with Lipiodol; we call it a "naked filling." The bronchi are pushed together and have a curled-up appearance (Figs. 3, 4, and 8). This resembles bronchiectasis, but after expansion of the lung the bronchogram is perfectly normal (Fig. 5). It is important to know this picture.

SUMMARY

A clinical picture is described, characterized by a temporary high position of the diaphragm. Various causes are pointed out; all patients had an inflammatory process near the diaphragm, situated either above or below the diaphragm. Perhaps one should speak of a diaphragmatitis. By means of bronchography it was made clear that the high position has a very restricted influence on the lung; only the basal parts collapse, and on the upper lobe no influence can be determined. This question is probably very important for the physiology and pathology of the lung.

TRANSPLEURAL MUSCLES

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IN THE course of a routine laboratory dissection, in a Negro man 60 years of age, a unique anomaly was found in the right pleural cavity. The anomaly consisted of two muscles lying in the oblique fissure of the right lung (Fig. 1). The medial of the two muscles arose from the sixth rib one inch from the vertebral column and inserted into the central tendon of the diaphragm just behind the caval opening. The lateral of the two muscles took origin from the sixth rib three inches from the vertebral column and inserted into the extreme lateral

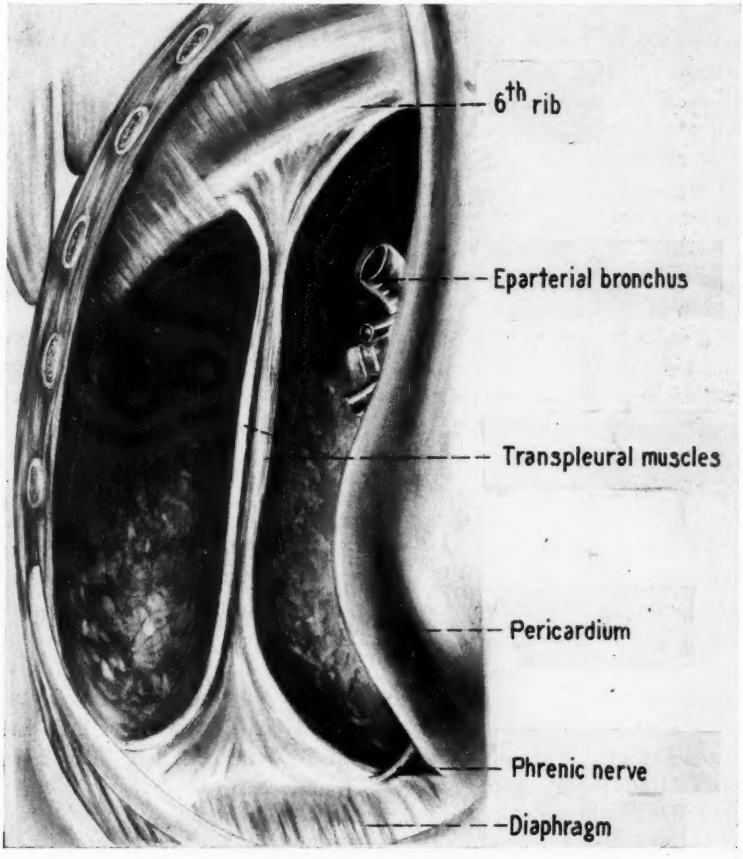

Fig. 1.—The superior and middle lobes of the right lung have been removed to show anomalous transpleural muscles in the oblique fissure.

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aspect of the central tendon. The muscles measured approximately six inches in length, their bellies making up about one-half the length. The bellies were slightly less than one-half inch in diameter. The tendons were thin and pearly and lay within well-defined sheaths which were condensation of loose fibrous tissue which covered the muscles and stretched between their tendons.

The origin of the nerve supply was not found, having been stripped away in the course of the student dissection. Stained section showed a preponderance of nerves in the lower portion of the muscles. This suggests that the nerve supply was phrenic rather than intercostal.

A careful search was made for associated anomalies but none were found. The possibility was considered that the fibrous sheath of the muscles was a part of an interlobar septum, but adhesions from an old pleuritic process made this impossible to determine.

It is of interest to speculate as to why similar anomalies have not been reported previously. Aside from its undoubted rarity we can only suggest that such a structure as described here might be mistaken, without close study, for an adhesion, as we first supposed it to be.

NONSPECIFIC GRANULOMATOUS (REGIONAL) ESOPHAGITIS

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THE intrathoracic causes of dysphagia in the adult usually fall into one of three main groups: cardiospasm, neoplasm, and stricture following swallowing of a corrosive. More recently interest has been taken in spasm or stricture associated with peptic ulceration of the esophagus.*

The three patients described here appear to fall into a different category and the investigations fail to show any association with tuberculosis, syphilis, or peptic ulceration, nor had any of them swallowed a corrosive. A feature common to all three patients appears to be the production of a nonspecific granulomatous lesion in the esophagus.

CASE REPORTS

Case 1.—C. M., a 52-year-old man, had a history of progressive dysphagia extending over a period of three months. The difficulty was first noticed with solid food and later even fluids became difficult to swallow. On one occasion he noticed blood in the regurgitated material. He was examined in India by esophagoscopy and the appearance left little doubt that he was suffering from a carcinoma of the middle of the esophagus.

The patient was flown to England on May 4, 1947, and because of the almost complete dysphagia, a jejunostomy was carried out before undertaking further investigation. As soon as his condition improved, an examination by barium swallow was made and showed an obstructive lesion at the level of the root of the lung and extending over a length of about two inches. The appearance was more that of a benign than of a malignant condition. In view of this x-ray finding it was decided to carry out cautious dilatation before proceeding to esophagoscopy in the hope that the value of this examination would be increased by this measure. Dilatation was carried out with gum-elastic bougies without difficulty, and up to Size 11 was used before there was any great resistance.

A roentgenogram made on May 31, 1947, showed an irregularity of contour in the middle of the esophagus with almost complete stenosis, and the appearance was most suggestive of malignant disease (Fig. 1). Esophagoscopy was made on June 4, 1947, and the instrument passed freely to 35 cm. where a hard swelling was seen occupying one quadrant of the esophageal wall, the lumen being displaced to the opposite quadrant. The swelling bled easily but did not appear to be fungating. A biopsy was done and showed granulation tissue heavily infiltrated with pus cells, and some squamous epithelium with signs of regeneration. There was no evidence of malignancy; results of the Wassermann and Kahn tests were negative. In view of the biopsy report, treatment was continued by autodilatation, the patient's condition rapidly improved and he was able to resume a normal diet. A second biopsy confirmed the findings of the first.

On July 1, 1947, another x-ray examination showed no irregularity in the outline of the esophagus. This was confirmed on Sept. 6, 1947.

Further progress was uninterrupted and the patient was eating normally by June, 1948.

Case 2.†—M. E. M., a well-developed girl 13 years of age was seen in April, 1942, complaining of increasing dysphagia. At first there was obstruction to solid food, but by the end of two months she was unable to swallow liquids and was rapidly losing weight (weight 85

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*Allison, P. R.: Peptic Ulcer of the Oesophagus, Thorax 3: 20, 1948.

†We are indebted to Professor C. Crafoord for permission to include the record of patient M. E. M., who was observed by one of us (S. T.) while working at the Sabbatsberg Hospital, Stockholm, as George Hunt Travelling Scholar of Oxford University.

pounds or 38.7 kilograms). The condition appeared to arise spontaneously. On examination she was thin but healthy, and x-ray examination May 12, 1942 (Fig. 2), showed an obstructive lesion starting in the middle of the esophagus. Above the stenosed part it was of normal caliber and contour and an esophageal bougie was held up 20 cm. from the teeth. In view of the patient's condition a gastrostomy was made on June 18, 1942, and her weight rapidly rose to 91 pounds (41.2 kilograms), with a corresponding improvement in general health. In February, 1943, her condition was very satisfactory, and she was readmitted for investigation. An x-ray examination showed an obstructive lesion at the level of the aortic arch. Esophagoscopy confirmed the presence of a stricture at this level and a fine bougie could be passed no further. Gastroscopy via the stoma revealed nothing of interest and repeated attempts at all types of bouginage failed, one being followed by a brisk mediastinitis which settled with sulfapyridine. The Mantoux reaction was negative even to a dilution of 1 in 30. By 1943 the patient was unable to swallow saliva, and it was decided to explore the esophagus.

Fig. 1 (Case 1).—Roentgenogram of barium swallow.

On June 1, 1943, the chest was opened* after removal of the seventh left rib. The lung appeared normal with a few adhesions. The esophagus was mobilized from the cardia upward; its walls felt hard and infiltrated except in the lower third which felt normal. At this stage a ureteric catheter was passed up from below but would not negotiate the stricture; similarly a sound introduced via an esophagoscope above was unsuccessful. The esophagus was divided at the level of the stricture below and the distal end invaginated. The gullet was then freed by blunt dissection and brought out through an incision in the neck. The operation caused little upset and the wound healed soundly. The esophagus lying in the dressings survived for one week and then necrosis set in rapidly to within 2 cm. of the stoma. Unfortunately no specimen was sent for section until this occurred.

*Surgeon, Dr. Crafoord.

The subsequent progress of the patient was uneventful. She continued to maintain her body weight satisfactorily by means of gastrostomy feedings and was able to attend school. The stoma in the neck contracted and required dilatation, suggesting that the original pathological process was still active, although it did not involve the pharynx.

Two and one-half years later, on Jan. 14, 1946, a biopsy was taken from the esophageal stump. The section showed no mucosa in the lumen, the wall being lined by granulation tissue of dense consistency. The muscle was replaced by fibrous tissue, and there was no sign of a neoplasm.

In December, 1947, the patient was attending school and in excellent health, and the first stage in constructing an antethoracic esophagus was carried out,* using Yudin's technique.

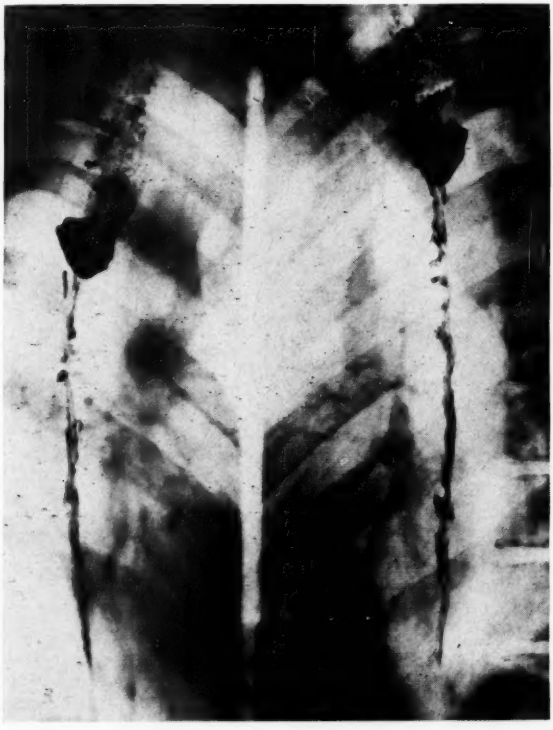

Fig. 2 (Case 2).—Roentgenogram of barium swallow.

CASE 3.—L. C., a single woman, aged 33 years, was referred to Hammersmith Hospital on Nov. 7, 1946, with a history of dysphagia which had started on Sept. 4, 1946, and had steadily progressed until it was almost complete.

The onset of the dysphagia was sudden and occurred at the end of a long illness associated with ante-partum eclampsia. The early weeks of the pregnancy had been uneventful, and apart from the mental anxiety associated with the circumstances of the pregnancy, she remained in good health until July 21, 1946, when she had a convulsion and was admitted to a hospital where she was found to have albuminuria and hypertension. Medical induction was started but was unsuccessful, and on July 25, 1946, hysterotomy with the removal of a dead fetus was carried out. She was about thirty-two weeks pregnant at the time.

*By Dr. Crafoord.

The patient herself could remember little of what happened between July 24, 1946, and Aug. 6, 1946, but it was stated that her temperature fluctuated at this time, although there was no gross sepsis. She received no blood transfusions at any time during this illness.

It was stated that her two married sisters, who had given birth to three children, suffered from eclampsia during each pregnancy.

Her temperature gradually settled and she was considered fit for discharge on Sept. 4, 1946. At breakfast on that morning she experienced pain on swallowing and this pain was repeated during the day. Solid food was regurgitated. The symptoms became worse, but she was able to take liquids easily until the end of September.

Fig. 3 (Case 3).—Roentgenogram of barium swallow.

When the patient was referred to Hammersmith Hospital, her weight had fallen from a normal of 131 pounds (59.5 kilograms) to 87 pounds (39 kilograms). She could swallow fluids with difficulty and solids not at all. X-ray examination showed an obstructive lesion at the middle of the esophagus (Fig. 3). It was considered that further investigations were not practicable until her condition improved and a Kader-Senn gastrostomy was carried out on Nov. 8, 1946. By Nov. 22, 1946, her weight had increased to 97 pounds (43.5 kilograms), and investigations were started to confirm the nature of her obstruction. Esophagoscopy* showed an ulcer encircling the esophageal wall at a distance of 25 cm. from the alveolar margin. A portion of the ulcer was removed for biopsy and was later reported to consist of granulation tissue heavily infiltrated with polymorphonuclear leucocytes. No sign of malignancy was found. The patient had a slight anemia and ocult

*By Mr. I. Griffiths.

blood was present in one out of three specimens of feces. Results from the Wassermann and Kahn tests were negative. The lungs showed no radiologic evidence of pulmonary tuberculosis, and although the sputum furnished doubtful tubercle bacilli on one occasion, this finding was not confirmed subsequently, and a guinea pig inoculation was negative.

Further radiologic studies were made through the gastrostomy opening and showed that the distal limit of the stricture was 2½ inches below the proximal block. No lesion of the stomach or duodenum could be demonstrated, and there was no evidence of pylorospasm. At this time a drip feeding was given at night through the gastrostomy, and some of this occasionally regurgitated upward through the esophagus. The gastric acidity was normal. Small size gum-elastic bougies were passed three times a day; passage was difficult and by Jan. 4, 1947, the esophagus accepted only size No. 7.

Because of the patient's slow progress, and the continuing suspicion of malignant disease, exploration was carried out through the right side of the chest on March 11, 1947 (R. H. F.). The stricture was localized at the level of the root of the lung and was about 1½ inches in length. Alongside the stricture was a lymph node ½ by ¼ inch in size; both gland and stricture being firm in consistence. The esophagus above the stricture was slightly dilated. The lymph node was removed for section. The esophagus was opened just above the stricture and the incision carried downward to healthy esophagus on the distal side. The lumen of the affected portions was almost obliterated and when found would barely admit a small probe. A portion of esophagus was removed for biopsy and esophagoplasty was carried out.

Convalescence was complicated by a right-sided empyema which necessitated drainage, but the patient was able to swallow comfortably until May 28, 1947, when she experienced a "spasm" and difficulty in swallowing, which was relieved by the daily passage of a Hurst mercury bougie. She was discharged on July 11, 1947, and advised to continue the use of the mercury bougie.

Bouginage was not carried out satisfactorily by the patient, and she was readmitted on Oct. 10, 1947, with a recurrence of dysphagia. With some difficulty it was found possible to introduce a ureteric catheter, but attempts to pass anything larger than this was unsuccessful. It was decided to re-establish the gastrostomy, recover the distal end of ureteric catheter from the stomach, and withdraw the catheter upward, having attached to its tip a fine rubber tube long enough to reach outside the mouth and abdominal wound simultaneously. This was done on Nov. 4, 1947. By means of the fine rubber tubing gum-elastic bougies were guided down the esophagus. A bougie (No. 13) was passed on Nov. 19, 1947. On Dec. 18, 1947, the gastrostomy tube was removed and the gastrostomy allowed to close. Dilatation was continued, but from time to time the patient complained of painful spasms during the passage of the bougies. On March 8, 1948, the patient started to menstruate, amenorrhea having been present since onset of the illness.

The patient found the use of a bougie intolerable and resection of the affected portion of esophagus was carried out in 1948 by one of us (R. H. F.) and continuity restored by intrathoracic esophagogastrostomy. Pathologic examination showed the condition to be similar to that found in Crohn's disease. The patient has since continued in good health and has had no trouble with swallowing. By using alkalies and avoiding lying flat, she has so far not had any evidence of peptic ulceration. However, in the light of later knowledge, it would have been more desirable to have restored the continuity by using small bowel instead of stomach.

DISCUSSION

The pathologic material from these three cases presented a nonspecific granulomatous condition of the esophagus. In Case 1 (C. M.) the clinical, radiologic, and esophagoscopic appearances all suggested a carcinoma of the

esophagus and if biopsy had been omitted and radiotherapy instituted, a cure by this treatment would have been claimed. The x-ray findings in the other two cases were strongly suggestive of neoplasm, even though both patients were young.

The cause of the condition is obscure; in two of the patients the onset of dysphagia was preceded by a period of mental stress. Geographically they were widely distributed: England, Sweden, and India. However, the histologic picture was similar in all three and one is tempted to compare it with regional ileitis.

The importance of this condition, which appears to be rare, is its clinical and radiologic mimicry of cancer. The diagnosis of esophageal carcinoma cannot be made without a histologic examination.

LOBECTOMY AND PNEUMONECTOMY IN THE TREATMENT OF PULMONARY TUBERCULOSIS

A Subsequent Report

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Boston, Mass.

IN MAY, 1946, a report* was made of the results of extirpative surgery applied in the treatment of 63 patients with pulmonary tuberculosis. This series was made up of 27 cases of lobectomy and 36 cases of pneumonectomy. The shortest period of follow-up in the series was one year and the longest was seven years (one case). The largest group was followed only two years (Fig. 1). At the time of this paper two years have elapsed since the last follow-up, and it is now possible to give the end results of all cases of the series after the expiration of three or more years from the date of operation. It would seem, therefore, that a subsequent report on the outcome of these cases is appropriate. This is particularly pertinent because a new factor in the form of prophylactic streptomycin therapy has been introduced since these cases were observed. It is probable that the immediate and possibly the late results will be improved because of the use of this antibiotic agent, but more time must elapse before its importance can be understood. The present series of cases, observed before the introduction of streptomycin, may assist in the establishment of a base line of the accomplishment of extirpative surgery alone, to which reference can be made in the evaluation of the assistance which streptomycin may provide in improving the results of treatment.

LOBECTOMY GROUP

The lobectomy group was comprised of 27 cases. There were 2 postoperative deaths, and by 1946 5 patients among those who survived the operation had already died as a result of the progression of the disease in the same or opposite lung. Thus there were 20 patients whose subsequent course can be studied. Of these 20 patients, 13 were apparently well at the time of the first report. Since then 6 of these have remained well continuously. Seven patients, however, developed exacerbations of the disease which required their readmission to the sanatorium for further treatment. In 3 patients the activity consisted of an exacerbation in the remainder of the lung on the operated side. Of these, 2 recovered and are now once again apparently well. The other patient is in only fair condition and is still under treatment in the hospital. One patient developed a flare-up in the opposite lung but has responded favorably to treatment and is now in an apparently well condition. One patient developed exacerbations of disease in both lungs. This patient's condition is poor at the present time and the prognosis is certainly unfavorable. Two patients developed bronchial ulcer-

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*Sweet, R. H.: J. Thoracic Surg. 15: 373-383, 1946.

ations. One of these has shown apparent complete clearing of the process. The other is slightly improved but is still in the sanatorium. These data are summarized in Table I.

Of the 20 patients still alive in 1946 after lobectomy performed one or more years previously, 7 were then suffering from manifestations of activity of the disease in the same or opposite lung. During the two years since that time 2 of these patients have improved to the point where they can be classified as apparently well. Three others remain unimproved and the other 2 have died. These facts are illustrated in Table II.

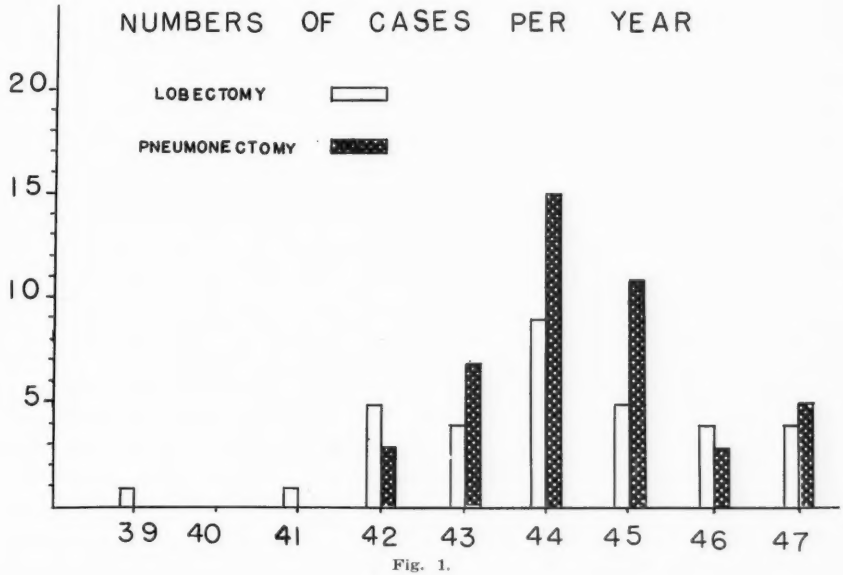

Fig. 1.

The revised end results of the original series of 27 cases are shown in Table III. Of the original 27 patients, 9 are dead. Of these, 2 died postoperatively and 7 died of late exacerbations of the tuberculosis. Of the 7 late deaths, 5 had occurred before the time of the first report and 2 occurred during the ensuing two years. Twelve are apparently well and 6 patients show some degree of activity at the present time.

PNEUMONECTOMY GROUP

The original pneumonectomy group consisted of 36 patients. Of these, 7 died postoperatively and 29 survived. But by the time the first report of results was made in 1946, 7 patients had died of the late effects of the progression of the disease. At that time also, 5 of the 22 patients still alive showed activity in the form of exacerbations in the opposite lung or of empyema on the operated side, which in the majority of cases developed late. In 2 patients these conditions

TABLE I. SUBSEQUENT COURSE OF THIRTEEN PATIENTS APPARENTLY WELL IN 1946 AFTER LOBECTOMY

CONDITION	NUMBER OF CASES	STATUS IN 1948 GOOD	FAIR	POOR	DIED
Remained well	6	6	0	0	0
Exacerbation in ipsilateral lung	3	2	1	0	0
Exacerbation in contralateral lung	1	1	0	0	0
Exacerbation in both lungs	1	0	0	1	0
Bronchial ulceration	2	1	1	0	0
Total	13	10	2	1	0

TABLE II. SUBSEQUENT COURSE OF SEVEN PATIENTS REPORTED IN 1946 AS ALIVE WITH ACTIVE DISEASE AFTER LOBECTOMY

STATUS IN 1948	NUMBER OF CASES
Apparently well	2
Not improved	3
Died	2
Total	7

TABLE III. END RESULTS OF LOBECTOMY FOR PULMONARY TUBERCULOSIS IN TWENTY-SEVEN CASES REPORTED IN 1946; REVISION AS OF 1948, THREE OR MORE YEARS AFTER OPERATION

Died		9
Postoperative deaths	2	
Subsequent deaths	7	
Alive with active disease		6
Apparently well		12
Total		27

coexisted. Seventeen patients were alive and apparently well. Table IV shows the subsequent course of these 17 patients. It will be seen from this that 12 of them remained well, but 5 developed subsequent difficulties. One sudden death from myocardial infarction, the result of coronary thrombosis, occurred. In the other 4 activation of disease in the remaining lung occurred in 2. One of these died. The other is apparently well at the present time. In one of the remaining 2 patients a small chest wall sinus developed in the incisional scar and in the other there was a flare-up of bronchial ulceration which has not yet entirely healed.

TABLE IV. SUBSEQUENT COURSE OF SEVENTEEN PATIENTS APPARENTLY WELL IN 1946 AFTER PNEUMONECTOMY

CONDITION	NUMBER OF CASES	STATUS IN 1948 GOOD	FAIR	POOR	DIED
Remained well	12	12	0	0	0
Activation other lung	2	1	0	0	1
Chest wall sinus	1	1	0	0	0
Bronchial disease	1	0	1	0	0
Coronary artery disease	1	0	0	0	1
Total	17	14	1	0	2

Table V shows what happened to the 5 patients who were alive but suffering from activity of the disease at the time of the first report. Two of these patients had empyema on the operated side and developed activity in the remaining lung

as well; both died. Two other patients had an empyema with fistula formation, one of the bronchus and the other into the esophagus. Both are somewhat improved as a result of thoracoplasty but not entirely well. The remaining patient succumbed to the effects of the disease in the opposite lung. Thus, of the 5 patients under discussion, none is well, 2 are alive with active disease, and 3 are dead.

TABLE V. SUBSEQUENT COURSE OF FIVE PATIENTS REPORTED IN 1946 AS ALIVE WITH ACTIVE DISEASE AFTER PNEUMONECTOMY

Apparently well	0
Somewhat improved after thoracoplasty	2
Dead	3

The revised end results of the series of 36 pneumonectomy cases are shown in Table VI. Of the original 36 patients, 19 are dead. Of these, 7 were reported previously as postoperative fatalities. Seven others had died from the late effects of the disease by the time the first report was completed. The 5 additional fatalities occurred during the ensuing two-year period of observation, making a total of 12 deaths from late activation of the disease. Three patients are alive but with evidence of activity. Fourtee· of the original group are apparently well.

TABLE VI. END RESULTS OF PNEUMONECTOMY FOR PULMONARY TUBERCULOSIS IN THIRTY-SIX CASES REPORTED IN 1946; REVISION AS OF 1948, THREE OR MORE YEARS AFTER OPERATION

Died		19
Postoperative deaths	7	
Subsequent deaths	12	
Alive with active disease		3
Apparently well		14
Total		36

A summary of the late results of extirpative surgery as applied in the treatment of 63 patients with pulmonary tuberculosis is given in Table VII. The duration of observation varies from a minimum of three years to a maximum of seven years after the operation was performed. The majority of the cases have been followed three and four years. It is apparent from Table VII that of the 27 patients who had a lobectomy performed, 9, or 33.3 per cent, are dead; 6, or 22.2 per cent, are alive with evidences of activity of the disease, and 12, or 44.4 per cent, are apparently well. In the pneumonectomy group of 36 patients, 19, or 52.8 per cent, are dead; 3, or 8.3 per cent, are alive with active disease,

TABLE VII. END RESULTS OF EXTIRPATIVE SURGERY IN 63 CASES (THREE- TO SEVEN-YEAR FOLLOW-UP)

	LOBECTOMY		PNEUMONECTOMY		TOTAL	
	NUMBER	PER CENT	NUMBER	PER CENT	NUMBER	PER CENT
Dead	9	33.3	19	52.8	28	44.4
Alive with active disease	6	22.2	3	8.3	9	14.3
Apparently well	12	44.4	14	38.9	26	41.3
Total	27		36		63	

and 14, or 38.9 per cent, are apparently well. In spite of the difference in the types of cases in each group there is a striking similarity of these figures if allowances are made for the greater postoperative mortality in the pneumonectomy group as compared with the lobectomy group (19.4 per cent versus 7.4 per cent).

DISCUSSION

Two additional years of observation of the series of patients reported in 1946 serve to bring into better focus the possible end results which can be expected after extirpative surgery in the treatment of pulmonary tuberculosis. It is very striking that of the 54 patients who survived the operation, only 19 have remained well continuously. Of these, 6 are of the lobectomy group (24 per cent of the 25 survivors) and 13 of the pneumonectomy group (45 per cent of the 29 survivors). One of the latter, however, died of heart disease. Thus, of the original 54 living patients, 35 developed subsequent reactivation of the disease after intervals of one to five or more years. Of these, 19 (76 per cent of the survivors) were in the lobectomy group and 16 (55 per cent of the survivors) were subjected to a pneumonectomy. It is obvious, therefore, that instead of the original statement that at least one-third had suffered a flare-up of activity following the surgical procedure, the present evidence suggests that approximately two-thirds (actually 75 per cent of the lobectomies) have manifested some form of late activity of persistent foci of disease. The reactivation consisted in almost all cases of flare-ups in the remaining homolateral or contralateral lung. In a few there was bronchial disease. The pneumonectomy group showed a striking incidence of late tuberculous empyema (4 cases, 2 with fistula).

Furthermore, although many of these patients improved subsequently, so that they may now be considered apparently well, 19 have died as a result of the progression of the disease (7 lobectomy and 12 pneumonectomy cases). It is more than ever apparent, therefore, that even though the major focus of disease can be excised surgically, any of the smaller remaining areas may at some future time become the site of activity of the infection. This serves to re-emphasize the importance of the immunologic aspect of tuberculosis and to stress the fact that this disease cannot be entirely eradicated by extirpative surgery applied to the lung.

It is an interesting observation that, as compared with those in the lobectomy group, the patients who had a pneumonectomy have fared rather better in so far as late results are concerned. Forty-five per cent of the survivors have remained well continuously as compared with 24 per cent of the lobectomy group. When the larger operative mortality is taken into account, however, there is relatively little difference in the over-all survival rates (Table VII).

It must not be assumed on the strength of these results that extirpative surgery should be discarded in the management of pulmonary tuberculosis. Reference to Fig. 1 will demonstrate that during the years 1946 and 1947 there were 16 resections performed, equally divided among lobectomies and pneumonectomies. The indications have been restricted somewhat, however, as a result of experience with the original series of cases reported upon first in 1946.

The indications for extirpative surgery at the present time are (1) irreversible bronchostenosis with chronic infection and secondary bronchiectatic dilatation, (2) lower lobe (or occasionally middle lobe) cavities, (3) persistent cavity after adequate thoracoplasty, and (4) possibly tuberculoma. Cases suitable for thoracoplasty are no longer considered for extirpation.

CONCLUSION

Subsequent observations of a group of patients with pulmonary tuberculosis treated by lobectomy or pneumonectomy serve to strengthen the original impression that extirpative surgery is a procedure of limited usefulness in the management of the disease. That such is the case is not surprising when proper consideration is given to the widespread dissemination of the infection which is present even at an early stage in almost all cases and to the fundamental importance of the immunologic balance between the host and the invading organism.

Whether the adjuvant prophylactic utilization of streptomycin may bring about an inhibition of the activity of the organisms sufficient to make extirpative surgery safer and more rewarding in its end results remains to be determined.

SUCCESSFUL REMOVAL OF LOWER ACCESSORY LUNG

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THE term accessory lung (aberrant or supernumerary lung) has been applied to several different types of pulmonary anomalies, of which tracheal lobes and so-called lower accessory lungs are most important. The tracheal lobe is located in the upper part of the chest and is usually connected with the trachea by an accessory bronchus, which probably represents a main and not a stem bronchus. The lower accessory lung, which is almost always situated at the base of the left lung, though it has occasionally been found below the left diaphragm, and which consists of sequestrated pulmonary tissue, is not connected with the bronchial tree. It would perhaps be better termed lower accessory lobe than lower accessory lung.

The case of lower accessory lobe reported in this communication is of interest because the anomaly is a medical curiosity; in the majority of recorded cases it has been an accidental finding at necropsy, and in this instance it was successfully removed at operation.

CASE REPORT

A 7-year-old Negro girl entered Charity Hospital at New Orleans on Sept. 16, 1946, because of a "tumor" of the lung. One month before admission she had been ill for three days with a headache, nonproductive cough, and a temperature of 101° F. Her physician made a roentgenogram of the chest and told her mother that she had a "tumor" of the lung.

Examination on admission revealed an apparently normal and well-developed child. A few slightly enlarged, soft, discrete lymph nodes were found in the cervical and inguinal regions. There was a soft systolic mitral murmur. The right lung was clear, but on the left side there was impaired resonance to dullness over the lower third of the chest posteriorly. The breath sounds were considerably decreased in this region, but there were no râles and no friction rub was audible. Fluoroscopic and roentgenographic examinations of the chest revealed a well-circumscribed, oval to round cystic mass above the left diaphragm posteriorly; the mass moved with respiration; the diaphragm was mobile (Figs. 1 and 2).

The impression was that the child had either a pulmonary or a dermoid cyst, and exploration was advised. Accordingly, on Oct. 1, 1946, the thorax was explored through a posterolateral approach, under endotracheal cyclopropane, oxygen and ether anesthesia The pleural cavity was opened by incision of the intercostal muscles in the sixth interspace. When the chest was opened, a large, smooth, somewhat rounded, brownish-gray soft mass was found lying posteriorly between the dome of the left diaphragm and the inferior surface of the left lower lobe of the lung. This mass, which measured approximately 8 cm. in diameter, was loosely adherent to the diaphragm and the lower lobe of the lung by areolar avascular tissue (Fig. 3). Elsewhere the surface, which was in contact with the thoracic wall, was smooth and glistening and resembled visceral pleura. The tumor was easily separated from the diaphragm below, and from the lower lobe of the lung above,

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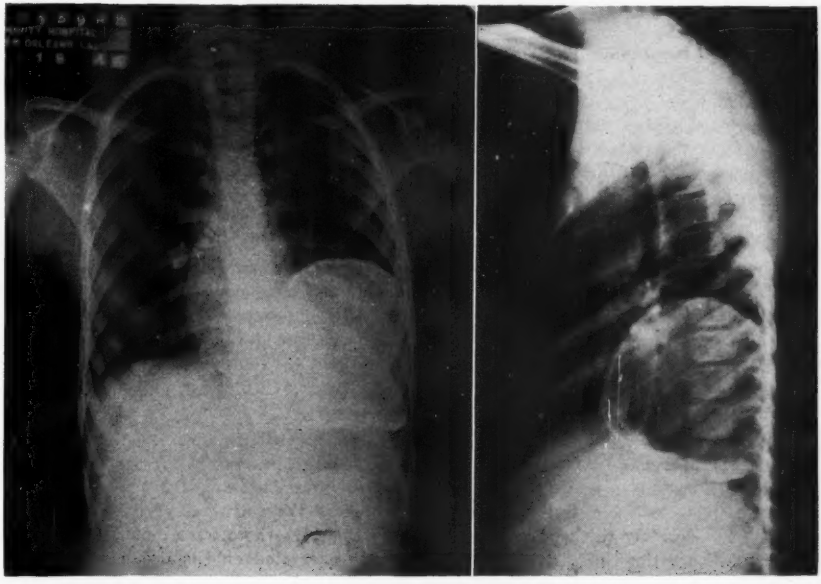

Fig. 1. Fig. 2.

Fig. 1.—Anteroposterior roentgenogram of chest in case of lower accessory lung showing well-circumscribed oval to round mass lying between base of left lung and diaphragm.

Fig. 2.—Lateral roentgenogram of chest showing well-circumscribed mass lying posteriorly just above diaphragm.

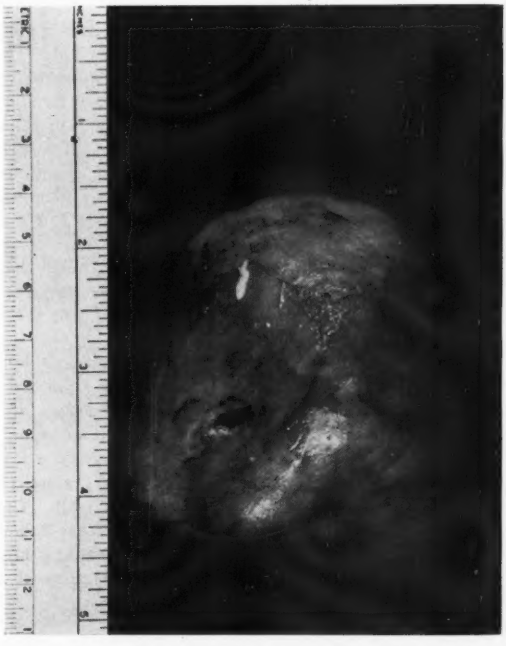

Fig. 3.—Lower accessory lung after surgical removal. Note irregularly spherical appearance and rather smooth surface.

by sharp and blunt dissection. After the mass had been freed from these structures, a small pedicle measuring 3.5 by 2 cm. was identified. The pedicle arose from the posterior mediastinum about 4 cm. above the diaphragm and well below the root of the left lung, which appeared grossly normal in all respects. The blood supply within the pedicle seemed to arise from the region between the esophagus and aorta, but the actual vascular communications were not definitely ascertained because it seemed unwise to prolong the operation by tedious dissection for this purpose. It was clear, however, that the arterial and venous supply of the tumor was systemic rather than pulmonic in origin. The pedicle was ligated and transfixed with quilting cotton sutures, and the mass was removed intact. The wound was closed in layers after the lung had been re-expanded by positive pressure.

The postoperative course was without incident and the patient was discharged from the hospital on Oct. 10, 1946.

In a follow-up report* on Nov. 17, 1947, fluoroscopic examination of the patient revealed "that the diaphragm was moving well, and that the lung appeared perfectly clear. In fact, except for the scar on the skin, it was difficult to tell that she had had any operative procedure."

Pathologic Report.†—The specimen consisted of an irregularly spherical fluctuant mass measuring approximately 8 cm. in diameter (Fig. 3). The external surface was smooth and gray except for a ragged area of brownish discoloration about 3 cm. in diameter. Minute vessels coursed over the surface. The bulk of the mass was occupied by a cyst filled with moderately thick, greenish-yellow fluid in which there were innumerable gritty particles. Concretions of similar appearance were encrusted on the lining of the cyst. In the areas free from concretions the lining of the cyst was yellowish gray and velvety. Several fibrous cords measuring up to 2 cm. in length and 0.1 cm. in diameter traversed the peripheral portion of the lumen of the cyst. The ends of these fibrous cords were attached to the cyst wall, but the cords themselves spanned small portions of the lumen of the cyst. The wall of the cyst was tough, gray, and fibrous and averaged 0.2 cm. in thickness, except at the points where the cords were attached; here it was thickened to as much as 0.5 cm. Within the thickened wall, at a point at which one of the fibrous cords was attached, there was a tiny cyst 0.1 cm. in diameter.

Sections were stained with hematoxylin and eosin, phosphotungstic acid hematoxylin, von Kossa's stain, Weigert's elastic tissue stain, and Ziehl-Neelsen's carbol fuchsin. In several areas the cyst was lined by pseudostratified columnar epithelium. Beneath the epithelium there was a distinct basement membrane five to ten microns in width. Beneath this membrane were lymphoid nodules with germinal centers, groups of mucous glands, and small numbers of elastic fibers and smooth muscle fibers. A number of circular to sausage-shaped plaques of hyaline cartilage were present beneath the lymphoid zone. The remainder of the cyst wall was made up of a thick layer of dense connective tissue fibers running roughly parallel to the lining of the cyst. Scattered through the fibrous wall were a number of nerves, large arteries, and veins. A large number of lymphoid aggregates of varying size were present throughout the connective tissue wall of the cyst; some of the larger aggregates appeared to represent actual lymph nodes.

In many areas the epithelial lining of the cyst was replaced by a thick layer of epithelioid cells throughout which were scattered foreign body giant cells and numerous large macrophages with foamy cytoplasm. Large irregular masses of calcium were incorporated in the wall of the cyst in these areas occurring predominantly in this inner epithelioid layer. The zone of epithelioid cells was surrounded by a layer of granulation tissue heavily infiltrated with small lymphocytes; this layer gradually merged with the dense connective tissue comprising the remainder of the cyst wall. The appearance suggested chronic inflammation and foreign body reaction rather than tuberculosis. No acid-fast bacilli could be demonstrated. There was pronounced intimal proliferation with narrowing or occlusion of the lumens of the arteries in the regions of extensive chronic inflammation.

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*We are indebted to Dr. Norman van Wezel for the report.

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Scattered through the wall of the cyst peripheral to the cartilaginous plaques were a number of small, irregular spaces lined by cuboidal or low columnar cells. The majority of these spaces were elongated and were about 140 microns in length and 70 microns in width, but others were circular. Their contours were usually smooth, but some had scalloped irregular outlines. These spaces resembled the epithelized alveoli seen in regions of chronic pulmonary fibrosis. They were usually arranged in groups of ten or less, the individual spaces in each group being separated by septa of fibrous tissue, elastic fibers, capillaries, and groups of small lymphocytes. Additional spaces of comparable size and shape, which had no epithelial lining, were separated from one another and from adjoining epithelized spaces by delicate septa composed almost entirely of capillaries. The majority of these spaces, including those with and those without epithelial linings, were empty, but a few contained large mononuclear cells, with abundant pink stippled cytoplasm, which resembled the so-called alveolar macrophages.

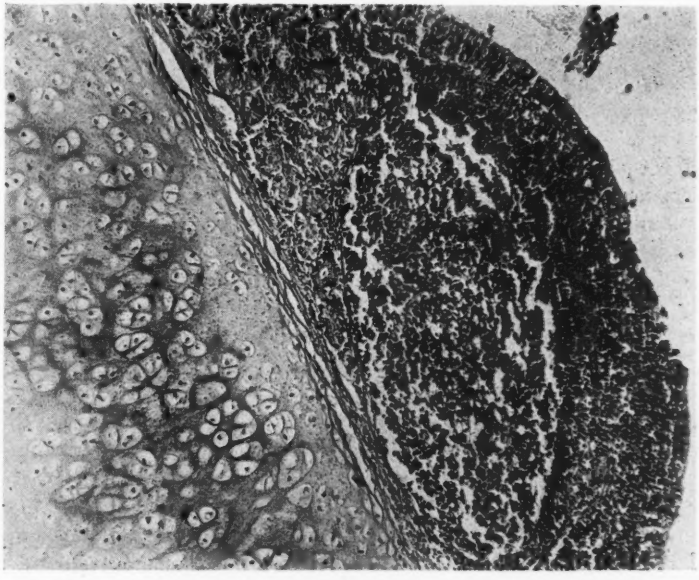

Fig. 4.—Photomicrograph of wall of cyst showing pseudostratified columnar epithelium, lymphoid aggregate, mucous glands, and hyaline cartilage.

The diagnosis of accessory lung was based on (1) the presence of a cyst lined by pseudostratified columnar epithelium and having a distinct basement membrane, mucous glands, and plaques of hyaline cartilage in its wall, and (2) the presence of epithelized and nonepithelized spaces separated by delicate vascular septa and having the appearance of pulmonary alveoli.

DISCUSSION

Lower accessory lobe is perhaps more common than the tracheal lobe, but it is still a rare condition. Since 1861 when Rektorzik and Rokitansky each reported a case, about forty instances in human beings have been recorded in the literature.[3-7, 9, 11, 12, 14-20, 23, 25-40, 42-46] These cases have been collected periodically and analyzed by various observers, including Bert and Fischer, Cockayne and Gladstone, Koontz, Scheidegger, Davies and Gunz, and Valle and White.

The majority of recorded cases of lower accessory lungs have been discovered at autopsy, often in infants who died at birth or soon afterward. Occasional cases have been reported in children by Quensel, von Mayenburg, and Humphry, and in adults by Vogel, Springer, and Freedlander and Gebauer. The latter authors, in 1939, successfully removed what they termed an "aberrant" lobe from a 21-year-old woman, but Valle and White, who apparently did not accept this case as authentic, in 1947, reported what they considered to be the first case of lower accessory lung discovered during the life of the patient and the sixth case in which the accessory pulmonary tissue occurred below the diaphragm. Their patient was a 9-month-old Negro boy who died, probably from shock, during operation for the repair of an associated diaphragmatic hernia. We believe our case is the first recorded of a lower accessory lung successfully removed at operation, as the case of Freedlander and Gebauer is excluded by most authors,[7, 43] presumably because of its atypical location and character.

Although numerous theories have been advanced to explain the origin of lower accessory lungs, an entirely convincing explanation of their derivation remains to be offered. Before any explanation of their derivation can be considered adequate, certain features of lower accessory lungs must be taken into consideration, including their frequent association with defects of the diaphragm, their vascular supply, which with rare exceptions is systemic rather than pulmonic, the absence of any bronchial communication between the accessory lung and the normal lung, and the preponderance of their occurrence on the left side. In only four of the forty cases that we have been able to collect, not including our own, was the accessory lung located on the right side. The distribution in cattle, in which the anomaly has also been reported, is more nearly equal on the right and left sides.[41] There are few commonly associated anomalies other than diaphragmatic hernia. The remainder of the lung is usually normal in position, size, and lobulation. The aberrant lung tissue in lower accessory lung seems to be notably free of disease or secondary infection, there being only one case[17] showing tuberculous involvement in association with tuberculosis of the normal lung and two cases with bronchopneumonia.[16, 25] This remarkable rarity of secondary infection is probably explained by the fact that few of the patients lived long enough for infection to develop and that the accessory lung tissue characteristically had neither bronchial nor vascular communication with the normally situated lung.

Two main theories have been advanced to explain the origin of lower accessory lungs, the theory of duplication and the theory of sequestration. The former was advocated by Eppinger and Schauenstein, who believed that these anomalies should be interpreted as true third lungs rather than as aberrant, sequestrated lung fractions or lobes. At the present time, however, such an origin from an accessory pulmonary anlage derived from the esophagus or stomach is not generally accepted[13] and the theory of sequestration is favored. This theory, which has been advocated by Vogel, Cockayne and Gladstone, Gruenfeld and Gray, and Davies and Gunz, attributes the aberrant pulmonary tissue to separation of a part of the primitive pulmonary anlage from the remainder

of the lung during embryonic life, with resultant formation of aberrant pulmonary tissue. Gross defects in the bronchial pattern of the normal, functional lung are occasionally found, but more frequently no such defects are described.[13]

The specimen from our case differs from many of those reported by others in the preponderance of bronchial elements and the relative scarcity of alveoli. Disproportionate development of bronchi has, however, been reported by others,[6, 7] and Freedlander and Gebauer stated that alveoli may be absent in lower accessory lungs. When alveoli are absent in a lower accessory lung, it may be impossible to distinguish it from a bronchial cyst in the mediastinum as in the third case reported by Freedlander and Gebauer. Bronchial cysts are more common in the region of the bifurcation of the trachea,[1, 2, 22, 24] and their walls may contain any of the tissues normally present in the trachea or bronchi.

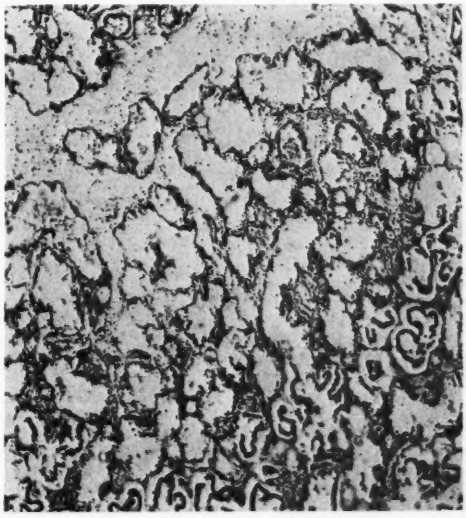

Fig. 5.—Lower accessory lung attached to the left leaf of the diaphragm in a stillborn infant. Note the interlobular septum composed of loose connective tissue and the alveolar-like spaces with and without epithelial linings.

SUMMARY

Lower accessory lung is an extremely uncommon anomaly which probably arises during embryonic life by separation of a part of the primitive pulmonary anlage from the remainder of the lung. In the case reported in this communication the accessory lung was successfully removed at operation.

ADDENDUM

Since the completion of this paper we have observed an additional example of a lower accessory lung. This occurred in a stillborn fetus weighing 4,220 grams whose death was apparently the result of rupture of a subcapsular hematoma of the liver.

On the superior surface of the left leaf of the diaphragm there was a sessile soft mass which measured 5 by 4 by 3.5 cm. This mass appeared to arise from the left leaf of the diaphragm, was not connected with the trachea, bronchi, lungs, or esophagus, and projected into the left pleural cavity. Its superior surface, which was smooth, was covered by diaphragmatic pleura. Its arterial supply was derived from a branch of the aorta arising just above the celiac axis and sending numerous branches through the diaphragm into the mass. The venous drainage of the mass was not determined. There was no defect in the diaphragm.

Histologic sections revealed innumerable spaces varying from approximately 100 microns in diameter to 1.0 by 1.5 mm. Many of the spaces contained desquamated columnar epithelium which appeared to have previously lined these spaces. Other spaces were lined by pseudostratified, columnar, ciliated epithelium and contained small plaques of cartilage and bundles of smooth muscle in their walls. Still other spaces had no epithelial lining, were separated from one another by delicate septa, and bore a striking resemblance to alveoli and alveolar ducts (Fig. 5). A few spaces contained a pale bluish material, but the majority were empty except for desquamated lining cells. The entire mass was divided into lobules by septa composed of loose connective tissue.

The appearance was that of pulmonary parenchyma in which large numbers of the respiratory spaces were lined by columnar epithelium.

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AN ARTIFICIAL AORTIC VALVE

Preliminary Report

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THE problem of restoring or improving the physical efficiency of incompetent cardiac valves has received little attention in surgical research, and there has been no report in the literature of any attempt to make a complete cardiac valve replacement.

In 1914, Alexis Carrel[1] reviewed his technique by which operations on the pulmonary and aortic orifices of the heart could be performed in dogs with some degree of safety. In one experiment he was able to cut the right pulmonary valve cusp with scissors and unite the laceration with fine silk suture. When the animal survived and retained a slight diastolic murmur, he reasoned and subsequently showed that "the bottom of the valve did not unite and a certain amount of insufficiency persisted."[2]

In 1938, Gordon Murray[3] resected with a valvulotome a portion of the lateral cusp of the mitral valve in dogs after the method of Cutler and Beck. He then took a segment of external jugular vein turned inside out, and threaded it through the anterior heart wall across the cavity of the heart to emerge on the posterior surface of the left ventricle. Anchored with slight tension, it made a free graft that bulged with systole into the opening left by the resected portion of valve. Cyanosis and cardiac dilatation were promptly controlled, and two dogs lived past ten days, remaining without signs of heart failure although they retained soft, blowing systolic murmurs.

The artificial aortic valve that forms the basis of this report is one of many valve patterns that have been fabricated from several types of plastic material. In 1937, Hufnagel[4] showed that a cylinder of methyl methacrylate approximately 4 cm. in length, 1 mm. in thickness, and with a diameter that was approximately the internal diameter of a canine aorta, could replace an excised segment of aorta, and that when several animals were sacrificed after six months there was found little tissue reaction at the junction of the intubation anastomosis and no mural thrombi were present.

Using the same methyl methacrylate, a tube with a central chamber was machined on a lathe (Fig. 1). Into the chamber was placed a moving valve machined and carved from the plastic. The length of the tube and contained valve is about 2.5 cm., thickness of the walls a little over 1 mm., and its over-all diameter about 1.2 cm. at the ends. This particular valve model opens and closes easily and quickly, and only 3 cm. water pressure is required to seat the valve (Fig. 2). The entire prosthesis is carefully polished on all surfaces.

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To produce aortic regurgitation in dogs, under Nembutal anesthesia the right carotid artery is exposed through a small incision in the anterior neck, and a semisharp stylet passed through the artery and aorta to aortic valve leaflets. The leaflets are deftly traumatized and aortic regurgitation is produced because of the thoroughly lacerated aortic valves. The trachea is then intubated and

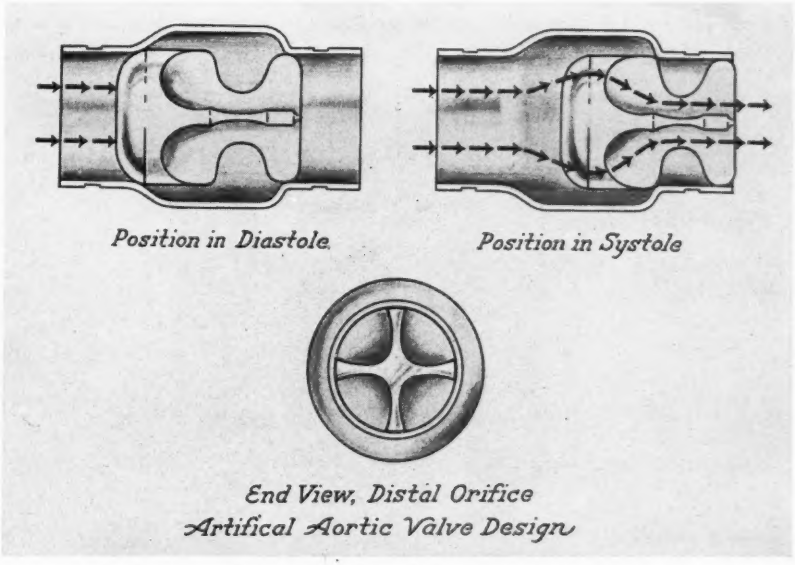

Fig. 1.—Diagram of artificial aortic valve.

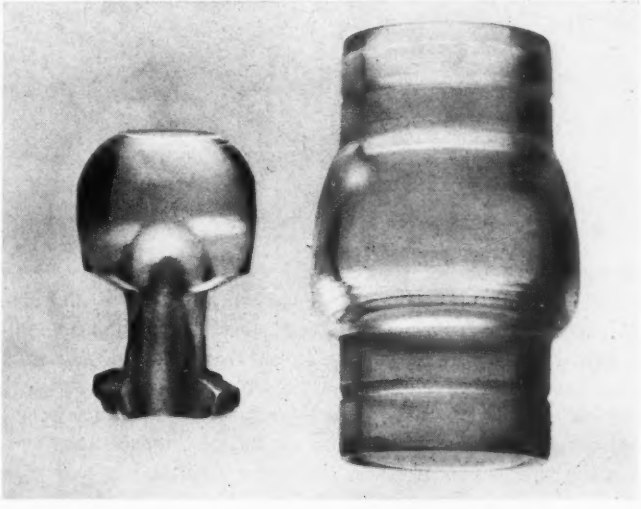

Fig. 2.—Component parts of artificial valve.

positive pressure respiration is begun. Next, a left anterolateral incision is made in the left fourth interspace, and the fourth costal cartilage cut. After packing away the left lung with moist gauze, and after all aortic branching vessels from the left subclavian to the fifth pair of intercostals are doubly ligated and divided, the aortic arch and descending thoracic aorta are thus mobilized. When completely free, the aorta is clamped at its proximal and distal dissected points, isolating from blood flow a segment of aorta about 10 cm. in length. A longitudinal opening now is cut in the vessel, the artificial valve inserted, the aortic

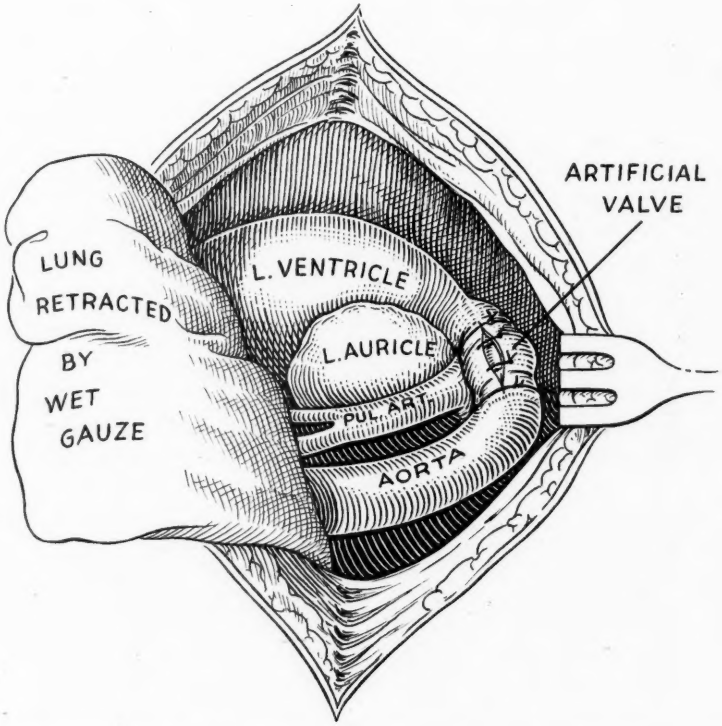

Fig. 3.—Position of artificial aortic valve. Exposure through fourth left interspace.

segment filled with citrate solution, and the valve anchored in place by means of No. 4 braided silk ligatures circling the aorta over each end of the valve chamber. The distal clamp on the aorta now is opened, then slowly the proximal one. The wound is closed with interrupted cotton sutures, and the collapsed lung re-expanded by aspirating the chest. The time interval during which the aorta is clamped off varies from 2¾ minutes to 4½ minutes. It also has been possible to close the opening in the aorta with fine silk sutures, release the encircling ligatures, move the valve upward across the aortic arch, and anchor it much closer to the aortic leaflets than shown in Fig. 3.

OBSERVATIONS

Kymographic recordings are made with a tambour connected to a femoral artery. After the aortic valves were lacerated, the normal pressure changed to one of aortic regurgitation. The systolic pressure rose, the diastolic pressure fell, and the resultant pulse pressure markedly increased within a matter of thirty seconds to five minutes. After placing the valve in the aortic arch, recordings of blood pressure approximated the dog's original pressure, although the shape of the curve varied from normal, due in part to the snapping action of the

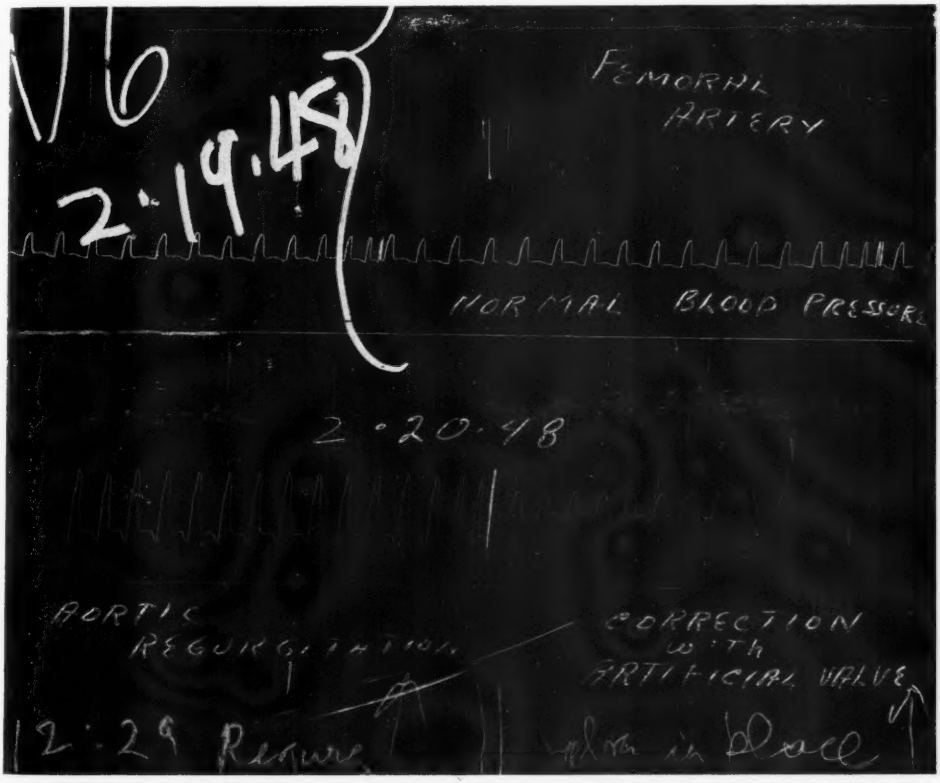

Fig. 4.—Femoral artery tracing. Normal blood pressure, aortic regurgitation, and corrected pressure with valve in place.

valve (Fig. 4). One tracing clearly showed that when open, minimal obstruction to aortic output was made by the valve or by the plastic chamber functionally replacing the aorta at its site.

A phonocardiographic record was kept of each dog. The normal heart sounds are replaced with the murmur of aortic regurgitation after tearing the aortic leaflets, and this murmur is replaced or overshadowed by the sound of the artificial valve (Fig. 5). Whether or not the valve is functioning can be easily

determined with a stethoscope, for when opening and closing it makes a sound resembling the ticking of an alarm clock. For some acoustical reason, probably the nearness of the valve prosthesis to the trachea, the ticking can easily be heard through the mouth of the dog when the jaws are held open, making a simple determination of whether the valve is in action and working from day to day.

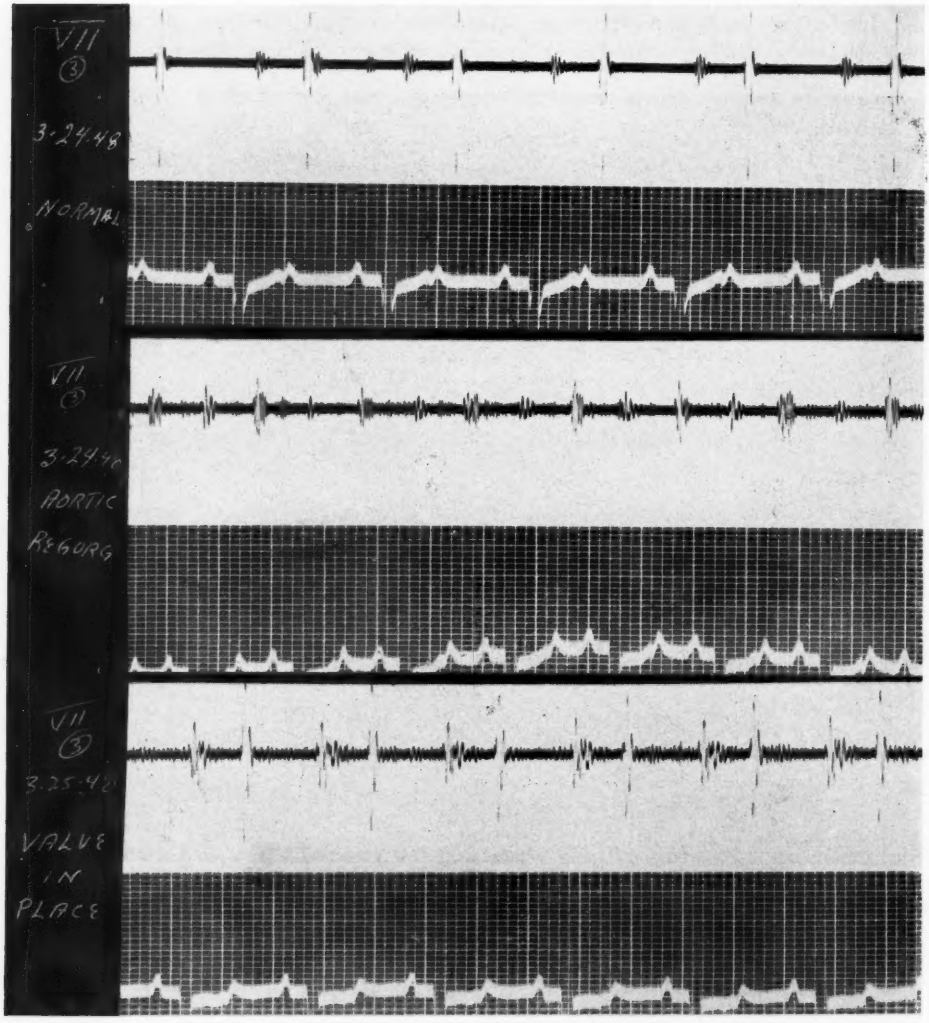

Fig. 5.—Phonocardiograph tracing of (1) normal aortic sounds, (2) aortic regurgitation, and (3) valve sounds, absence of aortic regurgitation.

With careful manipulation of the aorta, clotting of the valve at the site of its insertion has not been a problem. If, however, any traumatic ulceration of the aorta is produced while inserting the valve, thrombi are likely to develop in the

denuded areas. Anticoagulant drugs were not used in the group of dogs here reported.

In a series of five dogs in which this model valve has been employed, an elderly dog, weighing 20 kilograms, developed ventricular fibrillation twice while the aorta was clamped off, retained an arrhythmia that could not be controlled, and expired two hours postoperatively. One dog had a fatal hemorrhage on the fourth day, and the distal ligature about the valve was found to have cut through the aortic wall. Two dogs developed thrombi in the region of the lacerated aortic leaflets which dislodged on the third and fifth days and formed a plug in the valve that held it in the open position. Two dogs autopsied on the sixth and seventh postoperative days showed functioning valves, minimal tissue reaction, no evidence of thrombosis or technical difficulty. All of the dogs except the first one of this group were physically active from the first postoperative day. The ultimate survival is to be established by a series of dogs now under study and will be reported at a later date.

Although much remains to be done in these experiments, this report is made because it has been found possible to place an artificial valve in the circulatory system made of plastic substance and not autogenous tissue. With further improvement and study, it may be possible in the future to develop a valve prosthesis that may relieve aortic regurgitation in human beings, since it has been shown by these acute experiments that methyl methacrylate valves are tolerated in arterial blood flow and do not cause thrombosis as might be expected.

SUMMARY

1. Valves made of methyl methacrylate and placed within the aorta distal to lacerated aortic leaflets have been observed to function as a satisfactory correction of aortic regurgitation.

2. The valves have functioned up to seven days in this group of experiments.

3. Ultimate survival time is under study.

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DISCUSSION

DR. JOHN H. GIBBON, JR., Philadelphia.—Intracardiac surgery can be performed best by clamping both superior and inferior vena cava, and then operating in the open heart under direct vision. That is possible at the present time only for very brief periods of time. I think perhaps in the future if an extracorporeal circulation can be devised which will carry the circulation it may be possible to have the heart open and to carry out operations on septa and valves under direct vision. At the present time we are limited to short periods of about four minutes. Recently, Dr. Templeton in our laboratory has been able to clamp the vena cava, to open the right auricle, and under direct vision to excise one of the cusps of the tricuspid valve. He has replaced that defect in the tricuspid valve with a graft of vein or pericardium. The grafts of pericardium have been most successful. There are now five dogs living from one to five months after operation; two

of these show no murmur at all. We have had control dogs with cusps excised who show a definite murmur confirmed by phonocardiograms. I hope Dr. Templeton* may say a word about the technique.

DR. CLARENCE CRAFOORD, Stockholm.—I think that Dr. Templeton should be congratulated on the result of these experiments. The question that Dr. Gibbon raises is important for the further development of cardiac surgery. To develop a device for extracardiac circulation and extrapulmonary oxygenation in my opinion must be considered as essential for intracardiac operations. To be independent of the active breathing by performing controlled respiration I consider is now proved to be one of the prerequisites for major intrathoracic surgery. To make oneself independent of the cardiac action for the circulation I am certain will be as essential for intracardiac surgery. In my experimental department we have been working with a device that is quite different from the one Dr. Gibbon has constructed. We have now come so far that we can take over the circulation of the brain in a full-grown dog for as long as one and one-half hours, and then remove the artificial circulation and oxygenation machine and let the heart and the lungs take over their function again with surviving normal dogs.

In these experiments we introduced a cannula into the brachiocephalic artery, which provides the brain with blood, close to the aortic arch, after clamping this artery between the cannula and the arch. We clamped the inferior and superior vena cava, and tied the azygos vein. By this means the brain was adequately perfused during the experiment. The heart was bloodless because of the clamping of the cava. From my experience in cross clamping the aorta in a series of about forty cases of patent ductus I am convinced that if only the central nervous system is adequately perfused there will be a time of between twenty and thirty minutes for operations within an opened bloodless heart.

Editor's Note: Dr. Templeton's discussion was not returned by him. For that reason it must be omitted here.

SODIUM TETRADECYL SULFATE, AZOCHLORAMIDE, AND SULFANILAMIDE MIXTURE IN THE PREVENTION OF EMPYEMA FOLLOWING PNEUMONECTOMY WITH CONTAMINATION

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THE empyema that follows contamination of the pleural cavity after pneumonectomy for suppurative pulmonary disease constitutes one of the hazards in thoracic surgery. The lung to be removed is frequently adherent to the parietal pleura by adhesions formed by the underlying pulmonary pathologic conditions. In freeing an adherent lung from the chest wall, even though dissection is carried out in the extrapleural plane, a peripheral abscess is ruptured occasionally, and pus is spilled into the pleural cavity. A rupture into the cavity is more likely to occur when pulmonary resections are being done for pyogenic lung abscess and carcinoma complicated by a peripheral abscess; there is less likelihood of this rupture in resecting a lung for pulmonary tuberculosis or bronchiectasis. After division of the main bronchus, occasionally the clamp may slip from the bronchial stump, and infected bronchial secretions spilled into the pleural cavity may contain the organisms that instigate empyema. Empyema also may follow pneumonectomy where gross contamination is not apparent.

The opening of the bronchial stump with the resulting bronchopleural fistula is becoming a less common cause of empyema. However, when this type of empyema does occur, the infected pleural cavity is open to the outside through the tracheobronchial tree. This empyema is different from that caused by contamination of the pleural cavity at the time of operation when the bronchial stump remains closed. Drainage of the pleural cavity following total extirpations of the lung is almost certain to be followed by empyema if the drainage tube is allowed to remain longer than seventy-two hours.

Once empyema becomes established it is practically impossible to cure it by chemotherapy. Repeated aspirations and injections of chemotherapeutic and antibiotic agents usually will control the infection, but for a permanent cure the pleural cavity must be obliterated. This can be accomplished by drainage, and then allowing the space to become obliterated by elevation of the diaphragm, retraction of the interspaces, and overdistention of the remaining lung with a shift of the mediastinum to the affected side. Usually it is necessary to do an extensive thoracoplasty to accomplish the desired result. Strieder[9] reported that in his experience empyema promptly recognized and treated by extensive rib resection and open drainage is accompanied by an 85 per cent recovery rate. Temporizing measures, such as aspiration or closed drainage, result in higher mortality.

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In the past usually the pleural cavity was drained if there was evidence of contamination, and this procedure was followed with a thoracoplasty. Johnson[6] used this method and also observed that in a young patient with bronchiectasis the chest should be drained because of possible infection, and a thoracoplasty should be done to avoid future trouble from emphysema of the remaining lung.

Many combinations of chemotherapeutic agents and antibiotics have been used both locally and parenterally in order to eliminate empyema following intrathoracic surgery. Allbritten, Flick, and Gibbon[2] claimed a considerable reduction in empyema following the local application of sulfanilamide in the pleural cavity after lobectomy. White and associates[11] administered penicillin parenterally in a controlled series of patients who underwent resection (lobectomy and pneumonectomy) for suppurative lung disease. No other chemotherapeutic agents were employed, and penicillin was not used locally. They concluded that the occurrence of postoperative empyema in the controlled series is remarkable when compared to the absence of postoperative infections in those receiving penicillin. However, they reported, "It is our opinion that the preoperative administration of penicillin has been a most important feature of this study. The drug given during this period probably serves to control acute and chronic infection in the pulmonary tissues which reduces the possibilities of postoperative infections. In addition, the preoperative use of penicillin affords maximum therapeutic effect at the time the pleural space is exposed to bronchial contamination."

Blades[3] expressed the opinion that penicillin given preoperatively was not materially effective in preventing empyema in a small series of cases. Adams, Thornton, and Carlton[1] reported the use of 500 to 600 c.c. of blood plasma and 200,000 units of penicillin introduced into the pleural cavity following pneumonectomy in nine patients. They felt that the blood plasma reduced exudation and the loss of serum and tissue proteins, and that the penicillin was highly effective in preventing empyema.

It is noteworthy that no statistics could be found in the literature on the incidence of empyema following pneumonectomy, with or without contamination at the time of operation. When empyema[1-3, 5, 6, 9-11] was mentioned in the series of cases reviewed, it was listed as a common complication, but it was not stated whether or not the pleural cavity was known to have been contaminated at the time of operation.

The purpose of this paper is to report further results with a combination of drugs[4] which are believed to be especially effective in preventing empyema following gross contamination of the pleural cavity during operation. The first report concerned four patients who were treated with a mixture of sodium tetradecyl sulfate, azochloramide, and sulfanilamide. Although the first patient became infected, the treatment suggested the technique used in the succeeding cases, the results of which have been extremely successful.

Sodium tetradecyl sulfate is a wetting agent or detergent. By reducing surface tension it enhances the bactericidal action of many antiseptic solu-

tions. Petroff and Schain[8] found that it was most effective when used in combination with azochloramide. Neter[7] reported that the combined activity of sulfanilamide and azochloramide exerted a greater bactericidal action than when either was used separately. This suggested the possibility of combating the three drugs, azochloramide, sodium tetradecyl sulfate, and sulfanilamide.

When the pleural cavity is contaminated at the time of pneumonectomy, essentially the same procedure is carried out now as previously described.[4] Approximately 500 c.c. of the mixture containing azochloramide, 1:3,300, sodium tetradecyl sulfate, 1:500, and 5 Gm. of sulfanilamide are placed in the pleural cavity. The sulfanilamide crystals are inserted in the pleural cavity and then the solution of azochloramide and sodium tetradecyl sulfate is added. The chest is closed tightly without drainage. The intrapleural pressure is reduced to about 1 cm. of mercury negative pressure after the chest is closed. On three consecutive days after operation or until three consecutive negative pleural cultures are obtained, the pleural cavity is aspirated and the same mixture of three drugs is instilled. The quantity instilled depends upon many factors, including mechanical difficulty in aspirating and the necessity of leaving the pleural cavity under negative pressure after the aspiration and instillation. An endeavor is made to instill at least 200 c.c. and rarely more than 500 c.c. of the mixture at each treatment.

Including the first three cases reported, there have now occurred fourteen instances of grossly contaminated pleural spaces after pneumonectomy. These are consecutive cases and all patients have been treated by the method described. All have escaped infection or empyema, except the sixth one treated. This patient presented certain peculiarities that seem to warrant a detailed report.

CASE REPORT

A. S., a 46-year-old white woman, was admitted to the University Hospital on Dec. 9, 1947. In June, 1946, this patient had been diagnosed as having pulmonary tuberculosis. The third stage of a relaxing thoracoplasty was completed in April, 1947. Following this operation the tuberculosis was apparently arrested, and the patient was free of symptoms until several months prior to admission, when she developed a cough productive of a moderately large amount of sputum and a low grade fever. A bronchogram showed extensive bronchiectasis throughout the right lung under the thoracoplasty. Examination of the sputum and morning fasting gastric contents revealed no evidence of tubercle bacilli. On auscultation there was definite bronchial spasm bilaterally. The patient was moderately dyspneic. Pneumonectomy on the right side was advised and accepted. In preparation for the operation the administration of aerosol penicillin and streptomycin was prescribed. The patient became so dyspneic from bronchial spasm that she almost lost her life. Her reaction was almost as violent when the same antibiotics were injected. She was also found to be sensitive to the sulfonamides. On Dec. 29, 1947, a pneumonectomy was performed on the right side. The lung was freed from the chest wall with difficulty, and during manipulation it was noticed that there was spillage of a small amount of pus. Prior to closure of the chest the usual azochloramide, sodium tetradecyl sulfate, and sulfanilamide mixture was used. The regular three-day routine of aspiration and injection was carried out with the same mixture. Sulfanilamide was administered in spite of the previous reactions to it. In view of the danger of infection an antihistamin drug was given, followed by penicillin in streptomycin, but none of these could be tolerated. The culture taken at operation proved positive for hemolytic *Staphylococcus albus* and gamma Streptococcus. A culture of the pleural fluid on the first

postoperative day showed no bacteria. The temperature declined steadily to normal on the fifth day. It remained normal until the eighth day and then began to ascend. A culture of the pleural fluid on the tenth day showed hemolytic *Staph. albus.* It was evident that empyema was established. An open thoracotomy for drainage was done on the fourteenth postoperative day. The patient was discharged shortly thereafter and made an uneventful recovery. The pathologic report was bronchiectasis with cavitation.

The patient was sensitive to antibiotics and sulfanilamide. Streptomycin and penicillin caused such a violent bronchial spasm that neither could be used. However, the sulfanilamide placed in the pleural cavity was tolerated. From the culture reports one could reason that the mixture killed off the bacteria that originally contaminated the pleural space. A thoracotomy incision through an old thoracoplasty site is necessarily associated with a great deal of surgical trauma and a considerable amount of scar tissue that is often partially devitalized. The approximation of such tissue is not accurate when the wound is closed. Naturally, the chest wall is inoculated with the bacteria as well as the pleural cavity. The mixture placed in the pleural cavity cannot reach the bacteria in the tissues. It may be assumed that since the tissues were not aided by antibiotics, the bacteria created an infection in the chest wall tissues that spread to the pleural cavity on or about the eighth postoperative day.

The fourteen patients reported upon were given sulfanilamide by mouth for forty-eight hours before operation, and all received injections of penicillin or streptomycin, or both, after operation. Penicillin or streptomycin was not instilled into the pleural cavity.

It is thought that the mixture, when used as described will kill off all the bacteria that have been spilled into the pleural cavity at the time of operation. The mixture of azochloramide, sodium tetradecyl sulfate, and sulfanilamide is considered nonspecific in its bactericidal action. Therefore, it is much more desirable in treating a grossly contaminated pleural cavity than would be one of the antibiotics, such as penicillin or streptomycin, which is highly specific in its bactericidal action.

A rather extensive clinical trial of the mixture in treating pyogenic empyema has revealed that it will eliminate most types of pyogenic bacteria from the pleural cavity. After empyema has been established, the mixture will control but will not eliminate the hemolytic *Staph. albus* or *aureus.* It is hoped that it will eliminate these organisms if the attack against them is made before the organism actually invades the body tissues. It is reasonable to expect that contaminating bacteria would be more easily killed if attacked before they have had time to invade the body tissues.

The mixture placed in the pleural cavity can in no way influence bacteria contaminating the soft tissues of the chest wall. Therefore, it is extremely important that the patient receive the aid of penicillin and streptomycin in combating bacteria that contaminate the soft tissues of the chest wall. It is generally believed that penicillin and streptomycin will prevent infection from occurring in the soft tissue incisions. However, it should be realized that the parenteral administration of penicillin or streptomycin will reach the pleural fluid in small quantities.[10]

The objection to the preliminary report has not been overcome.[4] Complete bacteriologic studies are not available. In a surgical practice carried on in several different hospitals it is difficult to avoid the loss of specimens for culture, and under the stress of surgical work cultures sometimes are not taken.

Since the culture reports are favorable it is better to disregard these reports. In each case, however, pus was seen to spill into the pleural cavity. In every instance the pleural cavity and the wounds were closed without drainage. Only one patient out of fourteen developed empyema and that patient could not tolerate penicillin or streptomycin.

In eleven of the patients reported, the contamination occurred from an inadvertent tear into an abscess cavity. The bronchial stump clamp slipped from the divided bronchus in two cases, and the bronchial secretions which were assumed to be infected were seen to contaminate the pleural cavity. The postoperative pathologic diagnoses in the thirteen patients successfully treated were: carcinoma complicated by lung abscess, four; tuberculosis, six; bronchiectasis, two; and putrid lung abscess, one.

CONCLUSIONS

Sodium tetradecyl sulfate, 1:500, in azochloramide, 1:3,300, with sulfanilamide is an efficient nonspecific bactericidal agent.

When the mixture was properly applied to the pleural cavity after pneumonectomy with gross contamination, it prevented empyema in thirteen out of fourteen consecutive cases.

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THE MECHANISM OF HEALING OF LUNG TISSUE AND ITS REACTION TO DIFFERENT SUTURE MATERIALS

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LITTLE has been reported about the healing process of lung wounds or the reaction of pulmonary parenchyma to various suture materials. Olch and Ballon[4] in 1929 made linear incisions in the lungs of dogs and sutured them. One-half of the dogs received intratracheal injections of trypan blue after the operation. The dye was injected to determine the source of the scar tissue formation. It was found that the dye collected in pneumonic-like patches in the lung and that practically no pigment-bearing cells were found in the scar tissue. They reported that the scar produced in the lung, the result of a simple incision and suture, resembled a scar elsewhere in the body. More recently Montgomery[3] studied the healing of experimental wounds in the lung and the regenerative power of lung parenchyma. He cut a wedge, 3 cm. long on each side, out of the lung of adult cats. Bleeders, when encountered, were controlled by mattress sutures of silk, but he avoided the clamping and ligature of vessels as much as possible in order that the tissue might not be crushed. The visceral pleura was carefully approximated by interrupted silk stitches. The animals were killed at intervals of 1 to 112 days after operation. Histologic sections revealed well-formed bronchial buds at the apices of the wounds, which he thought indicated a regenerative phenomenon. He also reported the re-formation of the pleural membrane.

We have studied the reaction of the lung parenchyma of dogs to six different suture materials and their effects on healing.

METHODS

Thirty-two dogs were used in this experiment. Their average weight was eight kilograms. The sutures tried were catgut, chromic catgut, silk, linen, nylon, and cotton. The tubed nonabsorbable suture material was soaked in 95 per cent alcohol for about one hour and then autoclaved. Jenkins and Dunham[1] recommended this procedure because they found that tubing fluids often contained xylene and other highly irritating substances which cause considerable tissue reaction. The catgut was soaked in sterile water to make it pliable. The anesthesia used was intravenous nembutal in a dose of 30 mg. per kilogram of body weight. Positive pressure was maintained by an intratracheal catheter attached to a specially designed Foregger anesthesia apparatus. Sterile technique was used throughout.

In this set of experiments we carried out a technique identical with that used in pulmonary resection in man. The dog's chest was shaved and prepared with tincture of merthiolate. An incision approximately 10 cm. long was made along the fourth rib. The fourth rib was removed subperiosteally, and the pleural cavity opened. A lobe of the lung was brought to the outside. One or sometimes two Kelly clamps were placed near the periphery of the lobe and a slice about 2 cm. wide was removed distal to the clamp. A

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suture was tied at one end and a continuous stitch placed around the clamp or clamps. The clamp was removed and each stitch tightened individually before the other end was secured. A few bleeders were encountered, which were clamped and tied with the same suture material. The lung was then inflated with a pressure equal to 15 cm. of water, and the chest was closed in layers without drainage. Silk was used throughout in the chest closure. No dressing was applied.

Two operations were performed on most of the dogs, the second suture being placed in a different lobe. When the specimen was taken, the whole lobe was removed. The specimens were taken at intervals of 24, 48, and 72 hours, and 7, 14, 30 and 60 days.

We performed a second set of experiments in which no lung tissue was resected but the suture material was simply passed through the lung parenchyma. The suture material was threaded through a long needle, knotted at one end, and then knotted at the other end after being passed through the lung. The same studies were made on these specimens which were taken at 3, 7, and 14 days.

Several representative blocks of tissue were cut from each specimen. They were fixed in Zenker's fluid and imbedded in paraffin. Histologic sections were stained with hematoxylin and eosin, Masson's trichrome stain (after Goldner's modification), and Weigert's stain for elastic tissue.

WOUND HEALING

Repair in the pulmonary wounds proceeded in a characteristic manner.

Twenty-four Hours.—The pleural surface of the wound was covered by a thin layer of fibrin which extended over the hyperemic pleura for several millimeters on either side of the suture line. The cut edges of the pleura could be traced into the wound which appeared as a mass of hemorrhagic, necrotic tissue containing few recognizable alveoli and bronchioles. The pulmonary alveoli of the adjacent hyperemic lung were filled with blood. Small numbers of polymorphonuclear leucocytes were seen at the junction of the necrotic tissue and the blood-filled pulmonary alveoli. Small arteries and veins contained fresh thrombi, and some of the bronchioles and smaller bronchi contained a mixture of blood and fibrin in which only a few leucocytes were seen. There was no evidence of fibroblastic proliferation in any of the sections studied.

Forty-eight Hours.—The wound contained more polymorphonuclear leucocytes but was still bloody. The fibrous stroma in the adjacent normal lung appeared edematous. Bronchioles within the wound area and in the contiguous pulmonary tissue were lined by flattened epithelial cells. Some smaller bronchi were lined by two or three layers of polygonal cells which resembled closely those in the stratum germinativum of epidermis although no intercellular bridges could be seen. Mitotic figures were numerous among the cells lining the affected bronchi and bronchioles.

Seventy-two Hours.—Blood was less conspicuous in and about the wound. There was marked proliferation of all the fibroblasts in the stroma of the region. The alveolar walls of the adjacent lung were thickened. Apparently the peribronchial, perivascular, subpleural, and interlobular connective tissues all contributed to this response which extended for several millimeters into the normal lung. There was no leucocytic reaction in the proliferating areas and the leucocytes within the hemorrhagic and necrotic center of the wound were still polymorphonuclear. The epithelial cells lining the affected bronchi and bronchioles appeared to be squamous in type, but the lumina of these structures were well preserved. Fibroblasts had invaded the intrabronchial exudate, and organization was apparently proceeding without the presence of leucocytes.

Seven Days.—The core of the wound was now a mass of hemorrhagic exudate containing polymorphonuclear leucocytes, surrounded by a broad zone of vascular granulation tissue. There were moderate numbers of lymphocytes and plasma cells in some areas. No vestige of the damaged pulmonary alveoli remained, but there were focal accumulations of squamous epithelial cells surrounded by thin capsules which represented metaplastic bronchioles, and occasionally a typical bronchus was found lined by stratified squamous epithelium in the region of the wound (Fig. 1). There was much less blood in the wound

than formerly. The pleural splint was being organized by fibroblasts from the subpleural connective tissue, but the cut edges of the pleura could still be traced into the substance of the wound and their elastic fibers did not appear to be altered. A collagen-like substance was present in the thickened walls of the alveoli lying in the zone of fibroblastic proliferation seen at the end of 72 hours. Small perivascular accumulations of lymphocytes, plasma cells, and macrophages were seen in the lung tissue extending beyond the zone in which the alveolar walls were altered.

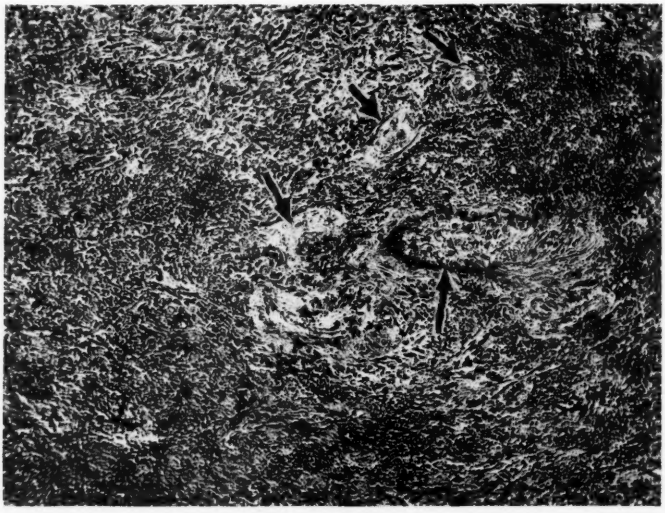

Fig. 1.—Squamous metaplasia of bronchiolar epithelium in healing wound of dog's lung 10 days after operation. (Hematoxylin and eosin stain, ×100.)

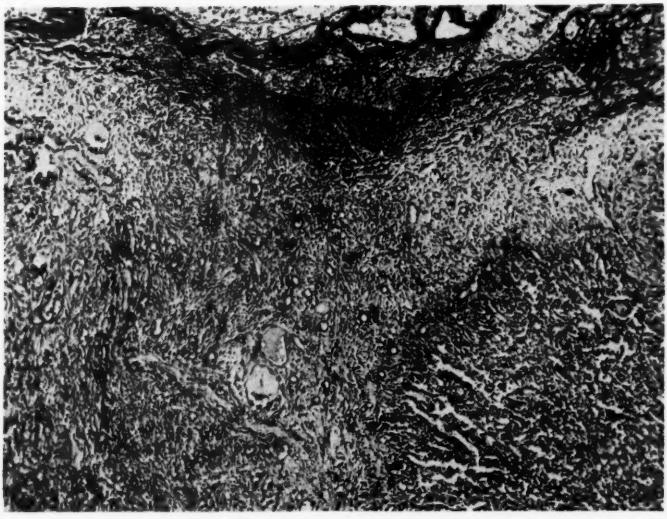

Fig. 2.—Healing wound of dog's lung 10 days after operation. Granulation tissue unites the cut edges of pulmonary tissue and an organizing fibrinous exudate still splints the pleural surface. (Hematoxylin and eosin stain, ×60.)

Fourteen Days.—Most of the wound area was a mass of young vascular scar tissue containing a few macrophages, lymphocytes, and plasma cells. Occasional bronchi were seen imbedded in the wound area. Most of them were lined by columnar epithelium, only a few by squamous cells. Some collapsed medium-sized blood vessels could be made out in the young scar tissue. The cut edges of the pleura could still be traced into the wound in sections stained to show elastica. The pleural splint was almost entirely organized (Fig. 2). A few small circumscribed foci of squamous epithelial cells and, occasionally, typical dilated bronchi lined by columnar cells were seen in the newly formed scar.

Thirty Days.—The wound area was considerably smaller and contained much collagen. The scar tissue was less vascular than at 14 days. Hyalinized remnants of bronchi and blood vessels and a few thin-walled bronchi filled with mucus and lined by columnar epithelium were imbedded in the scar tissue in several of the wounds studied (Fig. 3). The

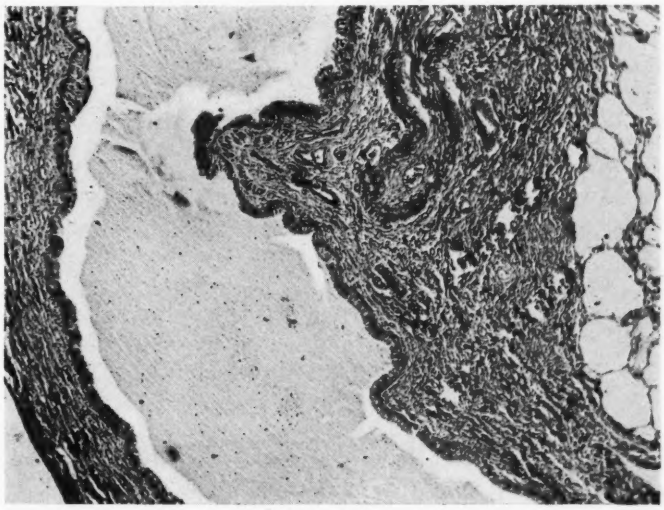

Fig. 3.—Healed wound of dog's lung 30 days after operation. Adult-type scar tissue has effected closure of the wound. The scar contains a trapped bronchiole filled with secretion and a thrombosed and recanalized artery. (Hematoxylin and eosin stain, ×60.)

elastica of the cut edges of the pleura was still visible. There was no evidence of regeneration of the pleura or its elastica in the depths of the wound. The pleural surface was covered by a layer of mesothelial cells. In one wound there were hemosiderin-laden macrophages about the scar, but generally hemosiderin deposits were not seen at any stage in the healing process. Strassman[5] has shown that blood injected into rabbit's lung may disappear rapidly without the local formation of hemosiderin.

INFLUENCE OF SUTURE MATERIAL ON HEALING

It should be remembered that the suture in each of these wounds had a dual function, apposition of cut surfaces and hemostasis. Each suture lay in fibrous tissue, usually peribronchial stroma. A zone of stroma and parenchyma 1 to 2 mm. wide between the suture and the wound was compressed as the result of the tension necessary for hemostasis (Fig. 4). This local reaction may have modified the healing process, but the assumption that any such effect of tension was relatively constant for all of the wounds studied seems to be warranted.

Wounds sutured with nylon appeared consistently larger and contained more histologic evidence of inflammatory reaction at all stages of development studied than did wounds closed with the other materials used. The nylon wounds still protruded above the pleural surfaces at one month when other wounds were depressed. The zone of hyperemia persisted

longer and was always wider. Considerably more scar tissue was formed, but there was no evidence that the process of collagen deposition was delayed.

The wounds closed with cotton and linen sutures contained more leucocytes than those in which silk or catgut was used. The inflammatory cells were still present in appreciable numbers at 30 days when cellular inflammation had almost completely disappeared from the other wounds. No incontrovertible evidence of delayed scar formation was found, however.

No essental differences could be seen in the histologic appearances of the wounds sutured with silk, plain catgut, or chromic catgut, save for a transitory rise in inflammatory reaction in the catgut wounds during the first week of the healing process. Absorption

Fig. 4.—Sutured wound of dog's lung 2 days after operation. Note general resemblance to infarct. (Hematoxylin and eosin stain.)

of the catgut led to isolated foci of foreign body and chronic inflammatory reaction about the suture area, but this process produced no notable change in the wounds.

REACTION ABOUT THE SUTURES IN THE WOUNDS

The tension on the sutures may have been responsible for some of the differences observed in the degree of inflammatory reaction about the suture material used to close the pulmonary wounds. The various suture materials were associated with the following reactions (Fig. 5):

Silk.—A few polymorphonuclear leucocytes appeared during the latter part of the first week, but most of them had disappeared at the end of the second week when there was some proliferation of the surrounding tissue. At the end of 30 days the suture was imbedded in a thin capsule of fibrous tissue, and no leucocytes were seen in the region.

Plain Catgut.—Polymorphonuclear leucocytes appeared about the suture during the third day and were moderately numerous at the end of a week. The sutures had been almost completely absorbed by the end of two weeks when the remnants were surrounded by foreign body giant cells, a few round cells, and a thin zone of fibrous tissue. One month after the wound was made, small fibrous nodules containing a few foreign body giant cells indicated where the catgut had been previously.

Chromic Catgut.—Small numbers of polymorphonuclear leucocytes were seen about the suture until the end of the second week when the catgut had become fragmented and was surrounded by foreign body giant cells, round cells, and proliferating fibroblasts. The

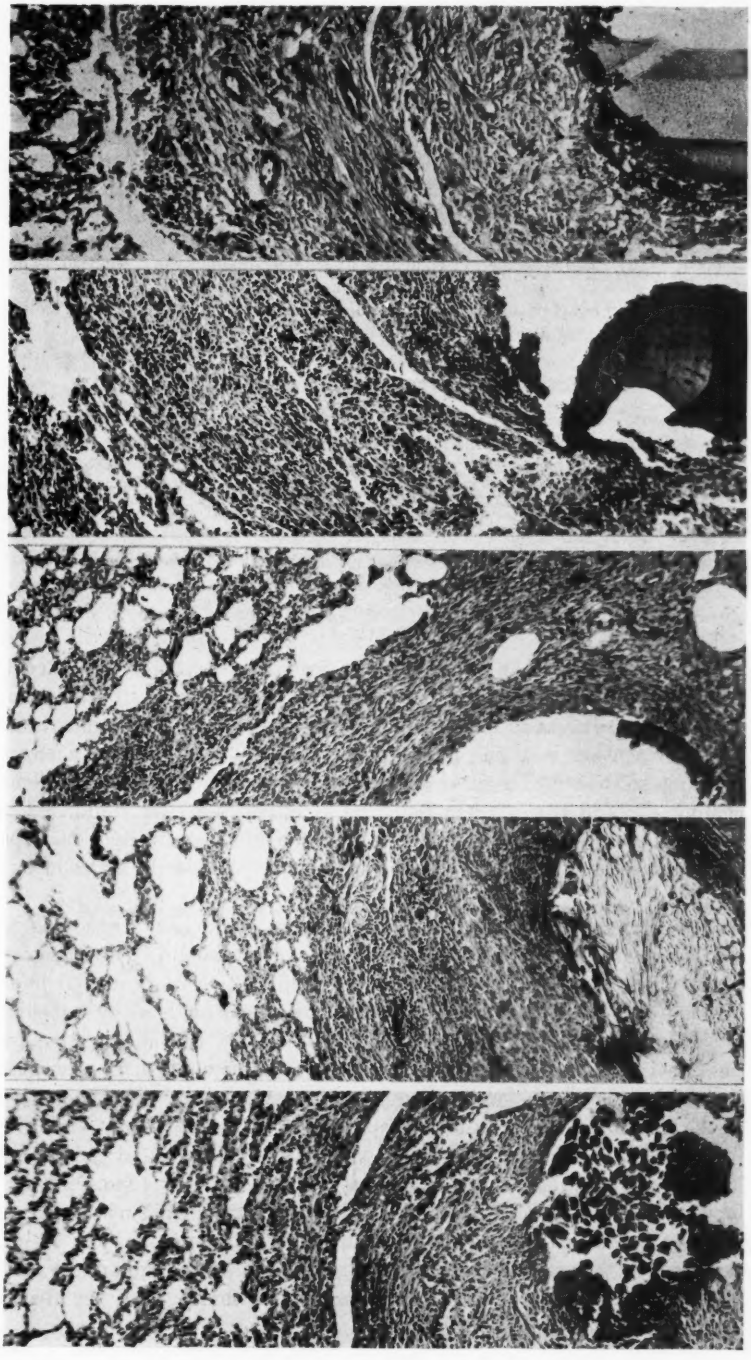

Fig. 5.—Tissue reaction about suture material used to close wounds of dog's lung 14 days previously. *A*, Silk; *B*, cotton; *C*, linen; *D*, chromic catgut; *E*, nylon.

fragments were smaller in specimens examined at 30 days, but the inflammatory reaction was similar.

Linen.—Many polymorphonuclear leucocytes were found in and about the suture material 24 hours after the wound was made. These cells persisted forming a dense collection about the linen so that the suture appeared to be in the center of a tiny abscess at the end of the first week. Thereafter the abscess-like lesion persisted and was walled off by fibrous tissue at the end of 30 days.

Cotton.—The changes were almost precisely the same as those described for linen.

Nylon.—At the end of 24 hours the nylon sutures were surrounded by a zone of necrosis as much as 100 microns wide. The degree of cellular inflammatory reaction found subsequently varied greatly. The constant change was a marked proliferation of fibrous tissue which enclosed the nylon in a fibrous nodule four to five times as large as the encapsulated silk suture at 30 days.

REACTION TO SUTURE MATERIAL ALONE

The various suture materials were imbedded in normal lung tissue by passing them approximately 2 cm. below the pleura and tying the ends loosely together over the pleural surface. In this way the reaction of the pulmonary components to the foreign bodies could be studied without the added effects that might have been due to closure of the wounds. Particular attention was paid to the nature and quantity of inflammatory exudate, fibroblastic proliferation, and deposition of collagen.

The changes observed about the silk, catgut, linen, cotton, and nylon were similar in all details to those described about the sutures in the healing wounds.

COMMENT

The technique employed in producing the wounds we studied produced a hemorrhagic necrotic mass which was organized by fibroblastic proliferation. The general principles seem to be the same as those described for wounds healing by second intention in other parts of the body. The structural changes observed have been described previously by Montgomery,[3] whose investigations on the healing of lung wounds are the most complete we have found. His procedure on cats was less drastic than the one used in these studies. Perhaps this may account for failure of the pleura to regenerate its elastic tissue in our dogs.

These studies lend no support to the thesis that pulmonary tissue regenerates itself in the process of repairing a parenchymal wound. Montgomery described the pneumatization of scar tissue by proliferation of bronchial buds and the formation of new alveoli. He assumed that spaces lined by cuboidal epithelial cells were newly formed bronchial buds. Cells resembling cuboidal epithelium line the thick-walled alveoli adjacent to the wound area after one week. The thickening of the walls is due to the appearance of a substance which has the staining characteristics of collagen but is not fibrillar. While the week-old wound contains many thick-walled alveoli lined by cuboidal cells, these become less numerous in the wounds studied at two weeks and one month. Only a few persist at two months. This suggests that the change in type of lining epithelium may be caused by an alteration in the environment of the lining cells occasioned by alteration in their supporting tissue. Apparently the collagen-like substance disappears as healing progresses, and most of the alveoli are reconstructed in their original form.

Montgomery stated that buds from the bronchi aerated the scar, indicating regeneration of pulmonary parenchyma. Our studies indicate that the chief mechanism by which the scar becomes smaller is through the contracture of scar tissue. Recognizable bronchioles and bronchi were more numerous within the wound during the first two weeks after operation than in the later stages of healing. Recognizable alveoli were seldom found in the scar. The reduction of the numbers of parenchymal units in the scar tissue militates against the thesis that aeration is accomplished by invasion of bronchial buds. It seems more probable that parenchymal remnants are trapped in the wound and become imbedded in scar tissue.

The suture material used had little influence on the rate of healing of the experimental pulmonary wounds in dogs. The end result was similar in all cases. Certain differences in the degree of inflammatory reaction and fibroblastic proliferation may be significant on theoretical grounds. If the operative field were not aseptic it is possible that the increased intensity of cellular inflammatory reaction due to the sum of the effects of the infecting agent and the suture material might be more conducive to necrosis. It is probable, however, that the necrotizing potentialities of the infecting organism are more important than the inflammatory component elicited by the suture material. Nylon, however, seems to us to be a poor substance to introduce into the lung. All of the wounds closed with nylon were much larger at all stages of healing than were those sutured with other materials. Therefore, these wounds might provide a better medium for the growth of bacteria. The excessive amount of scar tissue formed in the nylon wounds seems undesirable. Scar tissue in large quantities is essentially weak. This may be of little practical significance in the lung where physiologic stresses are usually of a low order of magnitude. It would seem unwise to use nylon when other readily available suture materials produce none of these theoretically objectionable changes.

The results of this work seem to indicate that the most desirable suture materials for lung tissue are silk, plain catgut, and chromic catgut.

Acknowledgment.—We wish to express our appreciation to Johnson & Johnson, New Brunswick, N. J., who furnished the Ethicon sutures used in this work.

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NOTICES

TUMOR REGISTRY COMMITTEE

The Tumor Registry Committee was unable to obtain the necessary assistance for the program it was instructed to carry out. Unwilling to appear empty-handed at the Denver meeting, the chairman will attempt to gather data for the more limited objective which he suggested at the New Orleans meeting: The relationship of interval and late results of operation to the cancerous lesion. Dr. Neuhof and Dr. Aufses, aided by a Research Fund grant from the Mount Sinai Hospital, propose to make an inquiry among all the members of the Association in a form which will be simple and not time-consuming. They anticipate sufficient material for a report at the April meeting at which time the Association may decide if further development of the method would be a useful function of the Tumor Registry. All members are urged to respond even if there are only single cases to report.

THE JAMES ALEXANDER MILLER FELLOWSHIP FOR RESEARCH IN TUBERCULOSIS

The New York Tuberculosis and Health Association announces that a fellowship will be available from July 1, 1950, to June 30, 1951. The fellowship is designed to support a qualified medical investigator who will devote full time to a research project with a definite bearing on tuberculosis. Assurance must be provided that the applicant will be acceptable in the laboratory or clinic of his choice and that he will be provided with the facilities necessary for the pursuit of the work. It is preferred that such laboratory or clinic be located in New York, N. Y.

The stipend will be $5,000.

Application forms will be supplied on request to the New York Tuberculosis and Health Association, 386 Fourth Avenue, New York 16, N. Y., and must be submitted in duplicate not later than March 1, 1950. Announcement of the award will be made in April, 1950.

BOARD OF THORACIC SURGERY

In June, 1949, the Secretary of the Board of Thoracic Surgery met with the American Board of Surgery, and the problem of those individuals who are neither members of The American Association for Thoracic Surgery nor diplomates of the American Board of Surgery was considered. The American Board of Surgery allowed a new group to be admitted to the Founders Group at the discretion of the Board of Thoracic Surgery. This group will be known as group "D" and will consist of other surgeons who are not members of The American Association for Thoracic Surgery or diplomates of the American Board of Surgery and whose training, because of the period in which it was taken, does not qualify them to become certified by the American Board of Surgery, but who may upon careful scrutiny and adequate evidence of proficiency be eligible for consideration as founder members.

ASSOCIATION MEETING TRANSPORTATION

The Burlington Railroad has offered to set aside some pullman space on the Denver Zephyr leaving Chicago 5 P.M., April 13, 1950, for our membership enroute to Denver. This space will be held by Mr. William Moloney, General Agent, Passenger Department, 105 W. Adams Street, 711-712 Bankers Building, Chicago 3, Ill. (phone Wabash 2-2345). Reservations may also be obtained from your nearest Burlington representative in principal cities throughout the United States. At the same time, if desired, return reservations may be made. The Burlington representative, Mr. A. G. Matthews, will be on hand at the meeting Friday, January 13, in Dr. Harper's office in the Republic Building.